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DEPARTMENTS

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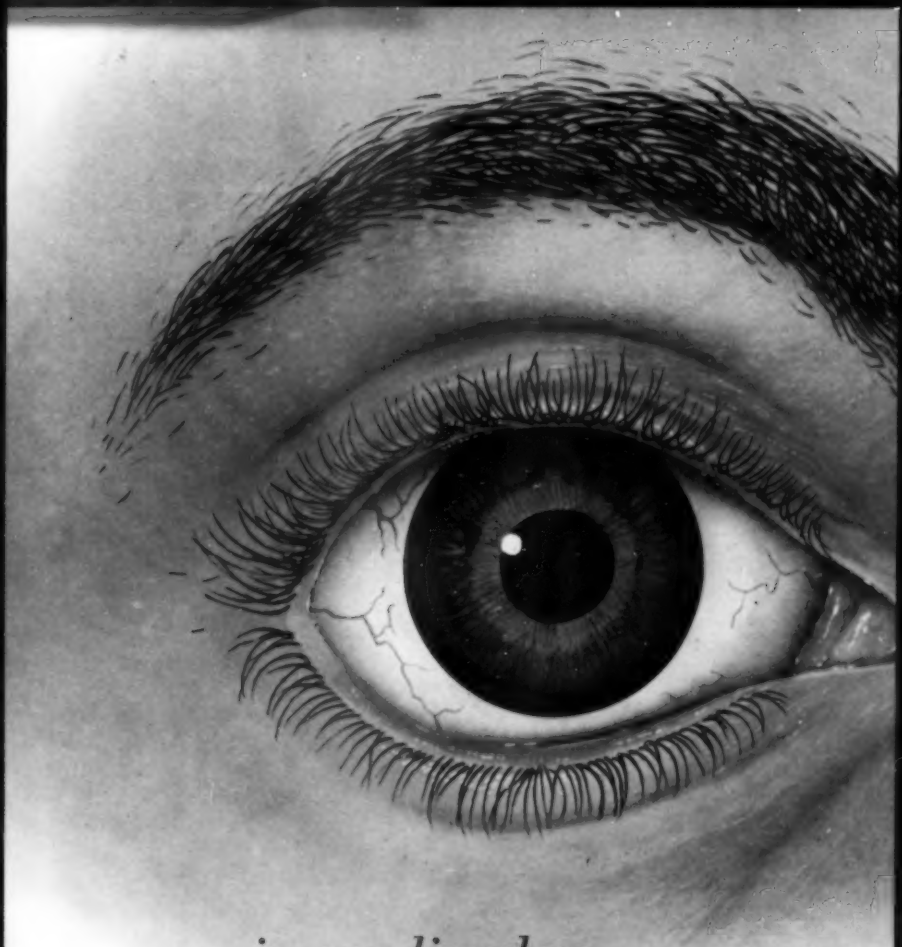

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AMERICAN JOURNAL OF OPHTHALMOLOGY

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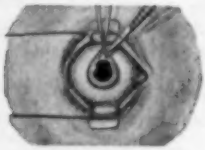

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 38 AUGUST, 1954 NUMBER 2

BLEPHAROPTOSIS: A GENERAL CONSIDERATION OF SURGICAL METHODS

WITH THE RESULTS IN 162 OPERATIONS

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The purpose of this communication is to present the results of a series of ptosis operations done by me or with my assistance at the Massachusetts Eye and Ear Infirmary during an approximate 10-year period. Pertinent embryologic and anatomic facts are considered, as well as surgical techniques, including my methods. Illustrated cases are also presented. Only those cases with adequate follow-up and personal supervision are included.

The total number of such operations reported herein is 162, and particular reference is made to a method of levator resection and advancement done through the skin rather than by the more popular conjunctival route.[1, 2]

These 162 operations were performed on 142 eyes of 113 patients. There are 129 levator resections, seven Dickey operations,[3–5] two Motais-Parinaud operations,[6, 7] three Berke's modification of the Motais-Parinaud procedure,[8] and 17 Friedenwald-Guyton frontalis suspension sutures.[9] The four remaining operations were canthoplasties done as a definitive ptosis procedure instead of as a supplement to a true ptosis operation.

The shortest postoperative follow-up in this series was two months, and the longest eight and one-half years.

In 99 of the operations, I was the surgeon; 16 are cases in which I assisted members of the Massachusetts Eye and Ear Infirmary surgical staff; and the balance are operative cases in which I was assistant to and instructor of Massachusetts Eye and Ear Infirmary ophthalmic residents.

A NOTE ON EMBRYOLOGY AND ANATOMY

Embryologically, the levator muscle is the last of the extraocular muscles to form. It starts to develop in the third month from the medial side of the superior rectus muscle[13] and most cases of congenital ptosis appear to be due primarily to improper development of the muscle itself rather than to improper development of its nerve supply. One of the strongest arguments in favor of this is the fact that the internal muscles of the eye are not affected in ordinary cases of ptosis, as would be expected if the condition were primarily due to involvement of the third nerve.

Due to the fact that the superior rectus develops first and the levator develops from it, the levator will always be absent if the superior rectus fails to develop, but the reverse is never true.

The anatomy of the orbit as it concerns ptosis surgery has been admirably described by Berke.[14] The levator muscle is flat, thin, shaped like an isosceles triangle, and originates at the apex of the orbit.

The levator tendon or aponeurosis originates from the levator muscle about 10 or 12 mm. above the upper border of the tarsus. It is closely applied to Mueller's muscle which lies on its under side and it cannot practicably be separated from Mueller's

muscle in operations for ptosis. Anteriorly it is covered by a thin areolar sheath which blends with the septum orbitale.

The aponeurosis extends forward and downward and is inserted chiefly into the orbicularis and skin in the region of the upper lid fold. Some fibers insert into the anterior surface of the tarsus. The underlying Mueller's muscle inserts into the upper border of the tarsus.

The aponeurosis has a thin medial horn which attaches to the internal canthal ligament and the posterior lacrimal crest, and a tougher, more fibrous lateral horn which attaches to the external canthal ligament and the lateral orbital tubercle. The lateral horn separates the lacrimal gland into two lobes.

It is the middle portion of the levator aponeurosis with the underlying Mueller's muscle which is utilized in levator resections. This will be further described in considering operative procedures.

An outline of the causes of ptosis

There are, of course, many causes of ptosis. True ptosis is due to insufficient power of the levator muscle because of an abnormality of the nerve supply or of the muscle itself. It may be unilateral or bilateral, partial or complete, and its origin may be congenital, acquired, or hereditary. It must be differentiated from pseudoptosis due to local disease such as trachoma, tumors, edema, enophthalmos, and so forth.

Acquired ptosis is the least common type. It may be due to:

1. Acute infectious diseases such as diphtheria, measles, meningitis, septicemia, and so forth.
2. Poisonings, as from coal tar or lead.
3. Syphilis, either local or involving nerve pathways.
4. Brain lesions, such as tumor, hemorrhage, and abscess (see Case 76, Table 2).
5. Other neurologic causes, such as myasthenia gravis, cerebral trauma, multiple neuritis, Horner's syndrome, and progressive ophthalmoplegia externa (see Cases 24, Table 1; Cases 32 and 53, Table 2; Case 67, Table 4).
6. Local orbital disease or injury (see Cases 17, 44, 64, 80, 103, Table 1; Cases 28, 35, 70, 72, 109, Table 2).
7. Hysteria.

Hereditary ptosis may be congenital or it may come on later in life. Rodin and Barkan[10] divide hereditary ptosis into four types:

1. Hereditary congenital ptosis (see Cases 20 and 27, Table 4).
2. Hereditary ptosis with external ophthalmoplegia.
3. Hereditary noncongenital ptosis (see Cases 8 and 29, Table 1).
4. Hereditary ptosis with epicanthus (see Case 54, Table 5; and Case 55, Table 4).

In ordinary congenital ptosis usually only one member of a family is affected but the hereditary type may affect several members; that is, it may be dominant or recessive, usually the former. Type 3 ordinarily occurs between 40 and 50 years of age, is a dominant characteristic, and is always bilateral.[11, 12]

By far the largest number of ptosis cases are congenital and nonhereditary and the bulk of my series is of this type.

Preoperative work-up in cases of ptosis

I. Investigation and handling of associated anomalies

Before attempting surgical correction in a case of ptosis, one must first look for the presence of associated anomalies. Some of these are:

A. *Abnormal motility of other extraocular muscles.* There may be strabismus of any type with or without frank paralysis of other muscles. It is, of course, most important to know whether or not the superior rectus muscle is functioning properly. If it is not normal, one cannot, of course, utilize it as a substitute for the levator.

In addition, it is important to note whether the eyes roll up normally when the lids are

forcibly closed; that is, Bell's phenomenon. If Bell's phenomenon is not present, there is some danger of exposure keratitis postoperatively.

Paralysis of the superior rectus is, of course, the commonest of the associated muscle anomalies. Two other conditions which may sometimes be found associated with ptosis are paralysis of upward gaze and third-nerve paralysis.

If strabismus is present, it should be corrected before proceeding with the ptosis operation. This is especially important when there is a hypotropia of the eye with ptosis because of the danger of postoperative exposure of the cornea.

B. *There also may be associated anomalies* of the lids, such as blepharophimosis (a fissure which is too short horizontally), epicanthus, or an abnormally long internal canthal ligament. If blepharophimosis is present, it should be corrected either before or at the time the ptosis operation is done.

The procedure which I use is the simple von Ammon canthoplasty (fig. 1). One first performs an external canthotomy. Through the cut edges of this wound, the upper and lower arms of the canthal ligament are cut by vertical incisions with scissors. The conjunctiva is then well undermined and sutured to the cut edges of the skin. This suturing must be quite accurate because there is a great tendency for the newly lengthened fissure to shorten after operation. This tendency to shorten can be minimized by sewing a thin piece of rubber tissue tightly against the new external canthus and leaving it in place for five days.

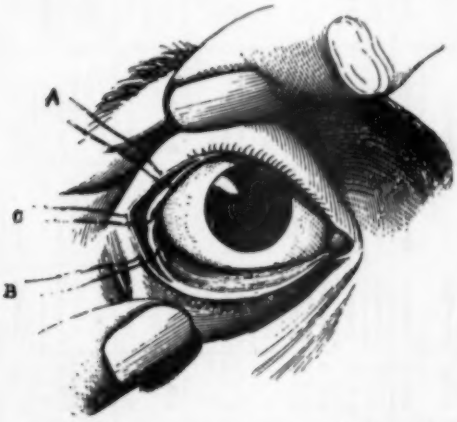


Fig. 1 (Johnson). *Von Ammon canthoplasty.* A, B, and C are silk sutures reapproximating skin and conjunctiva. The new external canthus is at (C) and it is here that a piece of rubber tissue may be sewn in temporarily to keep the new canthus open.

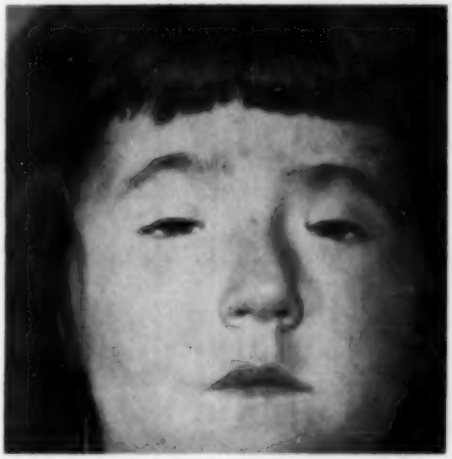


Fig. 2 (Johnson). *Epicanthus inversus* associated with ptosis and slight blepharophimosis.

The epicanthus seen in association with congenital ptosis is usually so-called epicanthus inversus (fig. 2). Actually the fold is more prominent below rather than above the internal canthus. It is frequently associated with blepharophimosis and a long internal canthal ligament, so that the internal canthi of the two eyes are far apart. This type of epicanthus does not change markedly with growth, so I usually prefer to correct it before proceeding with ptosis surgery.

The true epicanthal fold (fig. 3), most prominent above the inner canthus, is quite common in minor form in young children, is usually not seen in association with ptosis, and tends to become less prominent with growth.

The actual surgical treatment of epicanthus is beyond the scope of this paper.

TABLE 1

LEVATOR RESECTIONS UNCOMPLICATED BY OTHER MUSCLE ANOMALIES

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Amount Resected	Postoperative Complications	Length of Follow-up	Result	Remarks
								P.sop.	Postop.	Presop.	Postop.						
1	4	M	R	4/ 7 45	Congenital	None	None	2 mm. lower than O.S.	1 mm. lower than O.S.	N.S.	N.S.	Lev. resect.	7 mm.	None	4 mo.	G	
4	9	M	R	8/ 6/42	Congenital	None	Blaskovics	N.S.	N.S.	0	N.S.	Lev. resect.	"3 mm."	None	5 mo.	"G"	Examiner said "very good result"
5	11	F	L	6/ 5/45	Congenital	None	None	4 mm. lower than O.D.	1-2 mm. lower than O.D.	++	++	Lev. resect.	7 mm.	None	4 yr.	G	
6	8	M	L	2/21/49	Congenital	None	None	3 mm. lower than O.D.	Same as O.D.	N.S.	++	Lev. resect.	9 mm.	None	8 mo.	E	
7	8	M	R	11/18/47	Congenital	None	None	5.5 to 6.5	4.5	Lid fold 0	+	Lev. resect.	N.S.	None	5 yr.	F	Fair according to our criteria, but family very pleased
8	38	M	R	4/25/47	[? Trauma or hereditary both eyes but worse O.D.]	None	None	5.5	−2	+	+++	Lev. resect	8 mm.	Over-correction	2½ yr.	Over-corrected	Listed as fair
				6/27/47			Lev. resect.	−2	0	+++	+++	Lev. recess.		None	2½ yr.	F	Still very slight over-correction
9	30	F	R	3/22/46	Congenital	None	None	5.5	3	+++	+++	Lev. resect.	10 mm.	None	3 yr.	F	
10	11	F	L	3/ 6/51	Congenital	None	None	6 to 7	3	+	+++	Lev. resect.	10 mm.	None	13 mo.	E	
11	3½	M	L	10/16/52	Congenital	None	None	5	2.5	+	+++	Lev. resect.	17 mm.	None	5 mo.	E	
14	7	F	R	8/17/48	Congenital	None	None	4	2	++	+++	Lev. resect.	N.S.	None	4 yr.	E	
15	34	F	L	1/ 5/48	Congenital	None	None	4	1.5	+++	N.S.	Lev. resect.	12 mm.	Chalasion: (probably due to lid-margin suture)	10 mo.	G	Corrected to a point slightly *higher* than O.D.
16	5	M	R	8/ 2/51	Congenital	None	None	4	2	+++	++++	Lev. resect.	12 mm.	None	19 mo.	E	No detectable difference in height of lids in any position
17	3½	M	R	1/28/52	Orbital dermoid	None	Removal of dermoid	6	2	++	+++	Lev. resect.	10 mm.	None	17 mo.	E	On operating table it was noted that the lid appeared "too low" at end of operation
18	9	M	R	4/24/44	Congenital	None	None	5.5 mm. overlap	N.S.	0	N.S.	Lev. resect.	8 mm.	None	9 mo.	U	
				6/26/44	Congenital	None	Lev. resect.	5.5 mm. overlap	N.S.	N.S.	N.S.	Lev. resect.	4 mm.	None	7 mo.	E	Both lids exactly same height
			L	7/ 5/44	Congenital	None	None]	N.S.	N.S.	+	N.S.	Lev. resect.	N.S.	None	6 mo.	E	

TABLE 1—*Continued*

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Amount Resected	Postoperative Complications	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.						
19	37	F	R	3/ 7/44	Congenital	None	None	6 mm. overlap	N.S.	+	+	Lev. resect.	10 mm.	None	4 yr.	E	Both lids exactly same height
			L	3/ 7/44	Congenital	None	None	5 mm. overlap	N.S.	+	+	Lev. resect.	10 mm.	None	4 yr.	E	
22	2½	F	R	6/ 7/48	Congenital	None	None	N.S.	N.S.	?0	++	Lev. resect.	10 mm.	None	5 yr.	E	Examiner stated "O.D. is excellent, O.S. could be improved"
			L	6/ 7/48	Congenital	None	None	N.S.	N.S.	?0	++	Lev. resect.	15 mm.	None	5 yr.	F	
24	27	M	R	8/ 3/45	Acquired ? progressive ophthalmoplegia externa	None	None	5.5	0	++	++	Lev. resect.	10 mm.	None	3 mo.	G	Purposely overcorrected because this was a progressive condition
			L	8/ 3/45		None	None	6.5	−3	++	++	Lev. resect.	12 mm.	None	3 mo.	G	
29	47	F	R	3/16/48	Familial-acquired	None	None	4.5	−2	++++	++++	Lev. resect.	10 mm.	Over-correction	4 yr.	Over-corrected	Actually had slight ptosis left eye also; lid was at upper limbus immediately Postop. and went *higher* in next two months. (Listed as F.)
				8/ 5/48		None	Lev. resect.	−2	1	++++	++++	Lev. recess.		None	4 yr.	G	
31	4	F	R	2/19/42	Cong. O.U.	None	None	4.5	Same as O.S.	+	N.S.	Lev. resect.	7 mm.	None	6 mo.	G	Actually ptosis both eyes, but left pretty good, so op. only on right
34	15	F	R	6/30/45	Congenital	None	None	5.5	Same as O.S.	+++	+++	Lev. resect.	10 mm.	None	5 yr.	E	
36	14	F	R	7/10/50	Congenital	None	None	5.5 to 6	2.5	++	+++	Lev. resect.	N.S.	None	3 mo.	E	
37	5	M	R	6/14/48	Cong. O.U.	None	None	5.5	2.5	++	+++	Lev. resect.	5 mm.	None	4 yr.	E	Actually ptosis both eyes, but left not bad enough to require operation. Both same height. Postop.
38	4	F	L	12/27/51	Congenital	None	Blaskovics ? and F.-G. sling suture	6	2.5	++	+++	Lev. resect.	6 mm.	None	1 yr.	E	Previous operations done elsewhere, exact type not certain
39	13	F	L	5/ 4/46	Congenital	None	None	6.5	2.5	++	++++	Lev. resect.	8 mm.	None	6 yr.	E	
40	4	M	R	2/16/48	Congenital	None	None	5.5	3.5	+	+++	Lev. resect.	10 mm.	None	3 yr.	G	
			L	2/16/48	Congenital	None	None	7	3.5	+	+++	Lev. resect.	10 mm.	None	3 yr.	G	
43	5	F	L	12/14/51	Cong. O.U.	None	None	5.5 to 6	3	Lid fold 0	++	Lev. resect.	10 mm.	None	1 yr.	G	Actually ptosis both eyes. Final result = no difference between lids of the two eyes in primary position
44	24	M	R	8/18/42	Trauma—lid cut	None	Unknown type ptosis op. 6 yr. previous	6	N.S.	+	+++	Lev. resect.	5 mm.	None	2 mo.	G	

TABLE 1—*Continued*

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Amount Resected	Postoperative Complications	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.						
47	21	M	R	4/30/46	Cong. O.U.	None	None	6	3.5	+	+	Lev. resect.	7 mm.	None	2 mo.	E	Actually ptosis both eyes. Final result lids exactly same
48	3½	M	R	10/10/46	Congenital	None	None	10	3.5	+?	N.S.	Lev. resect.	12 mm.	None	11 mo.	G	Lids almost exactly same height in primary position, but some lag in upward gaze
50	19	F	L	9/ 7/50	Congenital	None	None	4	1.5	+	N.S.	Lev. resect.	N.S.	None	2 mo.	E	
51	5	M	L	8/25/48	Congenital	None	None	7.5	"Same as O.D."	0	N.S.	Lev. resect.	N.S.	Granuloma around one buried suture	7 mo.	G	
52	4	F	L	3/29/51	Congenital	None	None	5 to 6	4	+	+	Lev. resect.	12 mm.	None	2 mo.	F	
58	7	M	L	8/26/47	Congenital	None	None	6.5	3	0	+	Lev. resect.	10 mm.	None	5 mo.	G	Only ½ mm. difference in height of lids, but there is slight lag in downward gaze
60	7	M	R	9/18/45	Congenital	None	None	4	2	+++	+++	Lev. resect.	10 mm.	None	2 mo.	E	
62	4	M	R	5/22/46	Congenital	None	None	"4 mm. lower than O.S."	"Exactly same as O.S."	+	++	Lev. resect.	12 mm.	None	4 yr.	E	½–1 mm. overcorrected 4 mo. postop.
64	50	F	L	11/30/46	Trauma	None	Skin graft	6	2	++	+++	Lev. resect.	8 mm.	None	1½ yr.	G	Slight tenting of lid margin
				7/ 8/47		None	Lev. resect.	2	2	+++	+++	Repair of lid margin		None	1 yr.	G	
66	1½	M	L	9/18/44	Congenital	None	None	5.5	3.5	+	+++	Lev. resect.	N.S.	None	8½ yr.	F	Only ½ mm. lower than O.D. but there is excess skin which should be resected
68	65	F	L	4/12/46	Cong. and blepharo-chalasis O.U.	Blepharo-chalasis O.U.	Blaskovics	9	0	++	+++	Lev. resect.	8 mm.	None	3 mo.	E	
71	6	F	R	8/25/50	Cong. O.U.	None	None	5.5	3 to 3.5	+	++	Lev. resect.	10 mm.	None	2 yr.	G	Actually ptosis both eyes
73	16	F	L	8/16/51	Congenital	None	None	5.5	3	+	++	Lev. resect.	5 mm.	None	19 mo.	G	1 mm. lower than O.D.
74	32	M	R	10/16/51	Congenital	None	None	4	3	+++	+++	Lev. resect.	10 mm.	Very slight and almost symptomless keratitis	1 yr.	E	
75	22	M	L	6/ 7/51	Congenital	None	Some sort of levator op.—? tucking	7	2.5–3	++	++	Lev. resect.	10 mm.	None	7 mo.	G	

TABLE 1—*Continued*

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Amount Resected	Postoperative Complications	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.						
67	7	F	L	4/12/50	Congenital	None	Lev. resect.	"2 mm. lower than O.D."	"Same as O.D."	++	+++	Lev. resect.	6 mm.	None	2½ yr.	G	Not "E" because very slight tenting of lid margin
80	6	M	R	2/ 8/50	Trauma (dog bite)	Esotropia	Repair of laceration & attempted repair of levator	"Complete"	"Exactly same as O.S."	0	++++	Lev. resect.	10 mm.?	None	1 yr.	E	Really remarkable—No levator action preop. and postop. the lid appears practically normal
85	4	M	R	6/ 1/48	Congenital	None	None	N.S.	N.S.	+	+	Lev. resect.	8 mm.	None	4 yr.	G	
			L	6/ 1/48	Congenital	None	None	N.S.	N.S.	+	+	Lev. resect.	8 mm.	None	4 yr.	G	
86	18	M	R	7/11/42	Congenital	None	None	6	½ mm. higher than O.S.	N.S.	N.S.	Lev. resect.	6 mm.	None	2 mo.	E	
87	6	F	L	10/ 7/47	Congenital	None	None	5.5	1	++	++	Lev. resect.	N.S.	None	2 mo.	E	
88	5	M	R	6/16/45	Congenital	None	None	5	3	+	+	Lev. resect.	5 mm.	None	7 yr.	G	
			L	6/16/45	Congenital	None	None	6	3.5	+	+	Lev. resect.	7 mm.	None	7 yr.	G	
90	16	M	L	6/28/49	Congenital	None	None	6	1.5	+	+	Lev. resect.	12 mm.	None	2 mo.	E	
91	9	F	L	3/22/48	Congenital	None	None	"5 mm. lower than O.D."	3 to 3.5	?+	+	Lev. resect.	5 mm.	None	4½ yr.	G	
92	5	M	R	5/16/49	Congenital	None	None	4	3.5	++	N.S.	Lev. resect.	8 mm.	None	1 yr.	F	
			L	5/16/47	Congenital	None	None	3.5	2	++	N.S.	Lev. resect.	10 mm.	None	1 yr.	E	
93	6	F	L	4/12/51	Congenital	None	None	5 to 6	2 to 3	++	+++	Lev. resect.	10 mm.	None	1 yr.	E	
95	54	F	R	2/ 7/52	Recurrent erysipelas O.U.	Acquired Blepharophimosis O.U.	None	8 to 9	1	++	++++	Lev. resect. and canthoplasty	10 mm.	None	9 mo.	G	Really excellent result, but needs ptosis op. O.S. also
96	67	M	R	9/27/51	Cong. & Blepharochalasis	Acquired Blepharophimosis O.U.	None	9	2.5	Lid fold 0	+++	Lev. resect. and resect. of excess skin	15 mm.	None	1 yr.	E	Actually the only evidence of levator function preop. was slight deepening of lid-fold in looking up
			L	10/ 1/51		Acquired blepharophimosis O.U.	None	9	2.5	Lid fold 0	+++		15 mm.	None	1 yr.	E	
98	3½	M	R	12/15/50	Congenital	None	None	7.5 mm.	"2 mm. lower than O.S."	Lid fold 0	++	Lev. resect.	12 mm.	None	1 yr.	F	Only evidence of lev. action preop. was presence of lid fold, op. complicated by anaesthetic difficulties and had to be cut short
100	25	M	R	12/11/46	Congenital	None	Blaskovics	5.5	5	0	0	Lev. resect.	8 mm.	None	2 mo.	U	
			L	12/18/46	Congenital	None	Blaskovics	4.5	4.5	0	0	Lev. tucking		None	2 mo.	U	

TABLE 1—*Continued*

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Amount Resected	Postoperative Complications	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.						
102	7	M	R	8/ 1/44	Congenital	None	None	5.5	3.5	N.S.	N.S.	Lev. resect.	N.S.	None	2 mo.	G	
103	20	F	R	7/31/45	Birth injury	Absence of medial ⅓ of tarsus, lashes, and levator	None	5	"Medial ⅓ low"	?+	+	Lev. resect.	10 mm.	Redundant conjunctiva protruded under lid		F	Medial ⅓ of lid still low
				8/21/45	Birth injury	Absence of medial ⅓ of tarsus, lashes, and levator	Lev. resect.	"Medial ⅓ low"	"Same as O.S."	+	+	Resect. scar tissue of medial ⅓; resect. of redundant conjunctiva. Transplantation of lashes		None	1½ yr.	G	
104	4½	M	L	8/24/51	Congenital	None	None	5.5	3.5	++	++	Lev. resect.	10 mm.	None	1½ yr.	G	
106	5	F	R	9/24/41	Cong. O.U.	None	None	3.5	2.5	0	0	Lev. resect.	15 mm.	None	1½ yr.	E	Exactly same height as O.S.
107	16	F	R	7/29/42	Unknown acquired	None	None	5.5	"Very slight over-correction	+	N.S.	Lev. resect.	8 mm.	None	2 mo.	G	Intentional slight over-correction because condition possibly slowly progressive
108	17	F	R	10/10/50	Congenital	None	None	6	4	+	+	Lev. resect.	12 mm.	None	2½ yr.	F	
			L	10/10/50	Congenital	None	None	6	4	+	+	Lev. resect.	12 mm.	None	2½ yr.	F	
			R	5/ 1/52	Congenital	None	Lev. resect.	4	0	+	++	Lev. resect.	13 mm.	Transient exposure keratitis	1 yr.	G	Slight over-correction
			L	5/ 1/52	Congenital	None	Lev. resect.	4	1 to 1.5	+	++	Lev. resect.	13 mm.	None	1 yr.	E	
110	31	F	R	12/15/45	Congenital	None	None	4	1.5	++	++	Lev. resect.	7 mm.	None	6 mo.	E	
112	5	F	R	9/24/42	Congenital	None	Blaskovics	3 mm. lower than O.S.	5.5	+	++	Lev. resect.	6 mm.	None	8 yr.	F	Said to be "1 mm. over-corrected," 1 mo. postop.
113	71	F	R	5/16/52	Acquired	Blepharophimosis and blepharochalasis	None	5.5	0 to 0.5	+++	++++	Levator tuck skin excision	10 mm.	None	10 mo.	E	
			L	8/26/52	Acquired		None	3	0 to 0.5	+++	++++	Canthoplasty	10 mm.	None	7 mo.	E	

C. *An associated anomaly* which may sometimes escape notice because it may be very slight is the Marcus Gunn phenomenon, jaw-winking[19, 20] (fig. 4). Except in rare instances this affects only one eye and is most noticeable when the patient looks down and opens his jaw or moves it to the side opposite the affected eye. It is generally attributed to anomalous connection between the nuclei of the external pterygoid muscle (fifth nerve), and of the levator muscle (third nerve). It is somewhat akin to the acquired pseudo-Graefe phenomenon.[20-22]

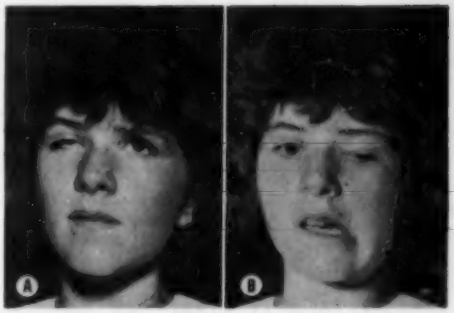


Fig. 4 (Johnson). *Marcus Gunn phenomenon of right upper lid.* (A) Ptosis of right upper lid with jaw closed. (B) The lid elevates when the jaw is opened and pushed to the side opposite the affected lid.

II. Preoperative measurements and investigation

In addition to a search for these associated anomalies, one must also make note of the following:

A. *The amount of ptosis* in the primary position and especially the difference in height of the two lids if the ptosis is unilateral. One should also note the height, or difference in height, when looking up and down.

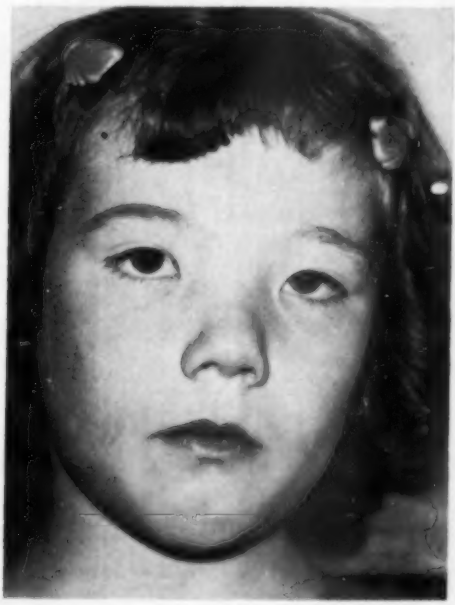


Fig. 3 (Johnson). True epicanthus in a young girl.

Accurate measurements of the amount of ptosis pre- and postoperatively are rather difficult to obtain. The amount of ptosis varies just as the height of a normal lid varies. In the case of the normal, this variation is slight, while in ptosis cases, it may be considerable, changing with the effort made to open the eye and with fatigue.

In the tables almost all measurements are recorded as "amount of overlap," that is, the amount that the lid overlaps the cornea in the primary position. This was measured with the brow held by the examiner's hand in those patients who used the brow to help elevate the lids. In some cases, this figure was not available so the width of the fissures or the difference between the two eyes is given.

Using the "amount of overlap" is a convenient and quick way to measure ptosis and is as accurate as any other method. The vertical corneal diameter in these measurements is considered to be 11 mm.

B. *The presence or absence of apparent levator action and the amount.*

In order to determine whether or not the levator is functioning, one must hold the brow, and then have the patient look up and down. If the brow is not held, the patient will elevate it in looking up, thus elevating the

TABLE 2

LEVATOR RESECTIONS IN EYES WITH OTHER MUSCLE ANOMALIES IN ADDITION TO PTOSIS

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Amount Resected	Postoperative Complications	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.						
2	16	M	R	12/21/51	Congenital	Weakness of upward gaze	None	6 mm.	3 mm.	+++	+++	Levator tucking	—	None	15 mo.	E	3 mm. overlap—both eyes same. Postop.
3	3½	F	R	9/ 4/52	Congenital	Slight jaw-winking	None	4 mm.	1 mm.	++++	++++	Lev. resect.	10 mm.	None	2 mo.	E	
12	5	M	R	6/ 1/49	Congenital	Weak sup. rectus	None	8 mm.	4 mm.	0	++	Lev. resect.	11 mm.	None	4 yr.	F	
23	10	M	R	2/25/46	Congenital	Paresis inferior oblique O.U., altecnating divergent strabismus	Resect. inf. oblique, recession ext. rectus both eyes	6	2	+	+++	Lev. resect. and recession sup. rectus	9 mm.	Wound bleeding	6 yr.	E	Corneal overlap 3 mo. postop. was 0; 2 & 6 yrs. later it was 2
			L	2/25/46	Congenital			6	2	+	+++	Lev. resect.	11 mm.	Wound bleeding	6 yr.	E	Corneal overlap 3 mo. postop. was 1 mm.; 2 & 6 yrs. later it was 2
29	9	M	R	1/12/51	Trauma anophthalmos	Has stone implant	Enucleation stone implant	N.S.	N.S.	++	++	Lev. resect.	N.S.	None	2 mo.	G	Height of lid adjusted by varying size of prosthesis
30	7	M	L	8/29/50	Congenital	Paresis sup. rectus, very marked jaw-winking	None	Varies from +7 to −2	Varies from +5 to −3	+++	+++	Lev. resect.	10 mm.	None	2 yr.	F	Variation in corneal overlap is, of course, due to jaw-winking
32	28	M	R	1/30/46	Progressive ophthalmoplegia externa	All extraocular muscles show almost complete paralysis; including orbicularis O.U.	None	5.5	4 to 4.5	0	0	Lev. resect.	15 mm.	None	6½ yr.	G	Listed as G; could not get lids any higher without almost surely losing both eyes from exposure keratitis. Orbicularis is extremely weak O.U.
			L	1/30/46			None	5.5	3.5 to 4	0	0	Lev. resect.	15 mm.	Exposure keratitis	6½ yr.	G	
33	6	M	L	10/ 9/52	Congenital	Marcus-Gunn phenomenon. Paresis sup. rectus	None	8.5 to 2.5	−2 to +3.5	++++	++++	Lev. resect.	20 mm.	None	7 mo.	F	Listed as fair, but family very pleased with result
35	40	F	R	2/ 3/48	Post-thyrotoxicosis	Weak sup. rectus and inf. oblique	None	6	3 to 3.5	+	+++	Lev. resect.	N.S.	None	4 yr.	G	
41	6	M	R	6/ 4/48	Congenital	Paresis III nerve	Previous Blaskovics allegedly made it "worse"	6	2 mm. lower than O.S.	+	+	Lev. resect.	10 mm.	None	4 yr.	F	Marked improvement but needs op. on vertical eye muscles
42	4½	M	R	7/11/47	Congenital	Paresis sup. rectus	On sup. and inf. rectus	4 mm. than O.S.	N.S.	++	++	Lev. resect.	7 mm.	None	6 yr.	G	
46	17	F	L	5/12/45	Congenital	III nerve paresis	None	N.S.	5.5	+	+	Lev. resect.	7 mm.	None	6 yr.	F	
			L	1/ 8/46	Congenital	III nerve paresis	Lev. resect.	5.5	5.5	+	+	Lev. resect.	5 mm.	None	5 yr.	U	
53	23	M	L	6/27/49	→	Prog. ophthalmoplegia externa	None	"4 mm. lower than O.D."	"1 mm. lower than O.D."	+	+	Lev. resect.	5 mm.	None	4 yr.	F	
57	6	M	L	7/19/42	Cong. O.U.	Weakness sup. rectus O.U.	None	N.S.	N.S.	+	N.S.	Lev. resect.	7 mm.	None	1 yr.	F	

TABLE 2—*Continued*

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Amount Resected	Postoperative Complications	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.						
61	3	F	R	9/19/47	Congenital	Epicanthus, blepharophimosis, paresis of sup. rectus	None	7	4	0	0	Lev. resect. and canthoplasty	14 mm.	None	5 yr.	F	
	4		L	3/ 1/48	Congenital	Epicanthus, blepharophimosis, paresis of sup. rectus	None	7	3.5	0	++	Lev. resect. and canthoplasty	N.S.	None	4 yr.	F	
	8		R	3/20/52	Congenital	Epicanthus, blepharophimosis, paresis of sup. rectus	Lev. resect.	4	3	0	+	Lev. resect.	14 mm.	None	2 mo.	G	
	8		L	3/20/52	Congenital	Epicanthus, blepharophimosis, paresis of sup. rectus	Lev. resect.	3.5	3	++	++	Lev. tucking	—	None	2 mo.	G	
63	16	M	L	8/20/51	Congenital	Jaw-winking	None	4	1	++	++	Lev. resect.	7 mm.	None	1 yr.	E	
65	8	M	R	1/28/48	Congenital	Jaw-winking and paresis sup. rectus	None	4 to 8	5.5 to 6	++	++	Lev. resect.	12 mm.	None	5 yr.	U	
70	17	F	R	8/23/51	Orbital tumor and removal	Paresis sup. oblique Paresis medial rectus Paresis inf. rectus	For tumor removal only	10	4	+ (outer ⅓ only)	++	Lev. resect. & myotomy inf. oblique	12 mm.	None	1½ yr.	F	Really quite good for this type of case
72	4½	M	R	1/25/49	Trauma	Anophthalmos stone implant	Stone implant	8	1 to 2	+	+	Lev. resect.	12 mm.	None	2 mo.	G	Height of lid can be varied by varying prosthesis size
76	20	F	L	8/28/47	Sclerosing fibroma of bone with complete III nerve paralysis and optic nerve atrophy		On E.O.M. only	"Complete"	2 mm. fissure	0	0	Lev. resect.	N.S.	None	2½ yr.	U	Really an unfavorable case for any type of operation
				2/ 2/48	Sclerosing fibroma of bone with complete III nerve paralysis and optic nerve atrophy		Lev. resect.	2 mm. fissure	3 mm. fissure	0	0	Lev. resect.	4 mm.	None	1½ yr.	U	
81	4	M	R	8/16/48	Congenital	Jaw-winking, weakness sup. rectus	None	5 (variable)	5 or less	+	+	Lev. resect.	14 mm.	None	2 yr.	U	
82	28	M	R	6/ 3/48	Cellulitis	Paresis sup. rectus	Drainage of cellulitis	6	3 mm. (same as O.S.)	0	0	Lev. resect.	12 mm.	None	1 yr.	G	Excellent result for this type case, but no levator action
89	6	M	R	8/28/44	Congenital	Paralysis of elevators	On E.O.M. only	N.S.	"3 mm. higher than preop."	+	+	Lev. resect.	8 mm.	None	6 yr.	F	Complicated by inervational muscle anomalies
94	3½	F	R	1/17/52	Congenital	Paralysis of upward gaze, nystagmus, divergent strabismus—alternating	Recession lat. recti and inf. recti; advancement sup. recti	See remarks	See remarks	0	0	Lev. resect.	12 mm.	None	1 yr.	G	Preop. the lids covered ⅓ of each pupil if she held her head far back. Postop. the pupils are uncovered without having to tip head back
			L	2/28/52	Congenital					0	0	Lev. resect.	12 mm.	None	1 yr.	G	
97	7	F	L	5/27/47	Congenital	Paresis of upward gaze	On vertical recti	5.5	3	0	0	Lev. resect.	8 mm.	None	5 yr.	G	Can close eye but it stays "wide open" in sleep—but never had any keratitis

TABLE 2—*Continued*

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Amount Resected	Postoperative Complication	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.						
101	15	M	R	11/12/45	Congenital	Paresis sup. rectus, ophthalmus	Repair of ophthalmus	4.5 to 5	2.5	++	++	Lev. resect.	10 mm.	None	2½ yr.	E	
			L	11/12/45	Congenital	Paresis sup. rectus, ophthalmus	Repair of ophthalmus	4.5 to 5	2	++	++	Lev. resect.	10 mm.	None	2½ yr.	E	
105	4	M	L	5/ 1/51	Congenital	Jaw-winking	None	4.5	2.5	+++	+++	Lev. resect.	12 mm.	None	2 yr.	G	
109	20	M	L	6/13/49	Tumor removal	Neurofibromatosis, paresis sup. rectus	Tumor removal Hom ptosis op.	Overlaps lower lid 5 mm.	5.5 to 6.5	?+	++	Lev. resect. and plastic repair	N.S.	None	2 yr.	F	Really tremendous improvement but still not a very good-looking lid. Had hearing aid; orbicularis spasm following each operation but no keratitis; result of each op. not maintained, apparently due to spasm
				11/ 7/49	Tumor removal	Neurofibromatosis, paresis sup. rectus	Tumor removal Hom ptosis op. and lev. resect.	5.5 to 6.5	5.5	++	++	Lev. resect.	N.S.	None	3 yr.	F	
111	9	M	R	10/14/49	Congenital	Paresis sup. rectus, exotropia	On vertical recti	5 to 9	3.5 to 4.5	+	++	Lev. resect.	11 mm.	None	1 yr.	F	

lid. This may give one an erroneous impression of the presence of levator function when in reality it is absent.

The presence of a lid fold *which deepens in upward gaze* indicates that the levator fibers which attach to the skin have at least some potential function. It has been my experience that when a levator resection is performed in the presence of such minimal evidence of levator function, the apparent levator action after operation is much greater than before operation. It is usually greater than may be accounted for by the theoretical presence of postoperative adhesions to the superior rectus or other structures which may move with the eyeball.

The finding of such a lid fold which deepens in upward gaze is enough evidence of potential levator function to warrant the performance of a levator resection, provided that the superior rectus is also reasonably strong.

For statistical purposes in this paper, however, levator function was graded according to the height of excursion of the lid. If the lid moved less than two mm. the levator was considered to have no action (0). Up to two mm. or so of movement may be obtained in perfectly flat lids without any lid fold, due to normal attachments between the levator and the superior rectus. In cases where less than two mm. motion was elicited but the lid fold deepened, a notation "lid fold" appears on the tables.

If the lid moved two to three mm. it was called 1-plus (+). If it moved three to five mm., 2-plus (++); and if more than this 3-plus (+++). Four-plus (++++) was used only in those cases in which the levator seemed to work nearly as well or as well as the fellow eye.

Some ptosis cases are seen in which there is no detectable difference in *action* between the levator of the ptotic and the fellow eye—these cases *appear* to have a levator which is simply too long.

Microscopic or electrical studies might in such cases show a difference, but clinically,

the muscle seems to *act* normally.* This is, of course, more often the case postoperatively in those who had fair or good levator function preoperatively.

C. *Another important consideration is the position of the upper lid fold of each eye,* so that one will be able to tell at operation where to make the new lid fold in the operated eye, in order that the two upper lids will appear symmetrical.

D. *One must also ascertain the vision, the presence of fusion, diplopia, or amblyopia.* If the eye is amblyopic and there is no fusion, one need have no fear of producing annoying diplopia, postoperatively. If diplopia is present when the lid is held open, one must correct this, if possible, before doing a ptosis operation.

If fusion is present, one must be careful that one's operation does not jeopardize it. This is especially important if one contemplates an operation utilizing the superior rectus to lift the lid.

E. *The sensitivity of the cornea must also be ascertained.* If one uncovers an anesthetic cornea, there is great danger of losing the eye through exposure keratitis.

III. When to operate

When to operate is a question of considerable importance.

A. *In cases of local lid trauma,* one may operate at any time after the damage, other than ptosis, has been repaired. Each of these cases is different and cannot possibly be considered here except in a general way.

B. *In acquired cases,* one may operate after one is sure that there will be no return of function or that medical treatment will not be effective, or that the patient does not have some progressive lesion. Some of the neurologic cases are poor surgical risks and some, like myasthenia gravis, respond to medical treatment.

In cases where operation is contraindicated, crutch glasses may be prescribed.

C. *In congenital cases,* if possible, one should wait until adequate co-operation can be obtained. This is about the same as in cases of strabismus.

If, however, a child has a *bilateral* ptosis which is so bad that he has to hold his head far back in order to see under the lids, one should operate at least as soon as he starts to walk.

If the ptosis is *unilateral* and *complete,* one should operate before amblyopia becomes irreversible. But if this eye shows a tendency to converge, one should operate much earlier.

If the ptosis is *incomplete,* and there is no amblyopia, one can and, I believe, should wait until about the age of three or four years.

Usually ptosis cases are best operated upon before the child goes to school and is exposed to the taunts of the other children.

Operative procedures

Once one has decided that a ptosis operation is necessary, one has a choice of three general types of operation.

I. Those utilizing the frontalis muscle for suspension of the lid.

II. Those in which the lid is attached to the superior rectus.

III. Those in which the levator is shortened or advanced, or both.

I. Operations utilizing the frontalis

These operations are of value chiefly in congenital cases, preferably bilateral, in which the ptosis is complete or nearly so, with no levator action, and in which the superior rectus is also weak or completely paralyzed; unless, of course, the frontalis is weak, which is quite rare.

The disadvantages are that the brow has to be elevated in order to lift the lid, sometimes the material used to suspend the tarsus from the frontalis stands out prominently in a band under the skin, and there is some danger of lagophthalmos. There is also a little more of an unnatural stare in down-

*I have been taking biopsies of the levator in cases of levator resection but the results of this investigation are not yet ready for publication.

ward gaze than there is after levator resection and considerably more than there is after suspension from the superior rectus.

The fact that the brow has to be elevated in order to elevate the lid is, of course, a disadvantage only in unilateral cases. A patient with bilateral ptosis without levator action usually uses his brow maximally in an effort to raise the lids. When this type of ptosis is unilateral he is less likely to do so. Use of the brow is necessary to the functioning of a frontalis suspension. Therefore, this type of operation is less often satisfactory in unilateral than in bilateral cases.

It has been stated that suspension from the frontalis is "an operation of fixation rather than of motion."[31] This is not strictly true. When the lid is suspended from the frontalis there should still be some degree of ptosis when the brow is not elevated; the ptosis is overcome by elevating the brow. If the ptosis is completely corrected with the brow in the relaxed position there will be dangerous lagophthalmos.

Various materials have been used to fasten the tarsus to the frontalis. They are: strips of skin,[29] silk or wire sutures,[9, 27] slings of orbicularis fibers,[26, 28] and slings of fascia lata.[30] The most satisfactory materials are autogenous fascia lata or orbicularis muscle fibers.

It is the Reese operation,[26] utilizing orbicularis fibers, which I have recently come to favor when the frontalis muscle is to be used to suspend the lid. However, in this series, all the frontalis suspension operations are patterned after the procedure described by Friedenwald and Guyton[9] which is a relatively simple and efficient operation.

The following description has been shortened and very slightly modified from that given by Friedenwald and Guyton.

Two stab wounds are made near the inner and outer ends of the brow, as shown in Figure 5. They go down to the periosteum. Two incisions in the skin of the lid are shown two mm. above the lid margin in this picture from Friedenwald and Guyton's article. I prefer to have them five or six mm. above the margin; that is, about in the position of a normal lid fold.

The suture enters the upper nasal incision, goes through the frontalis fascia, across and out the upper lateral incision. From here it goes down under the orbicularis and out the lateral lid incision. A bite is taken in the tarsus, then it goes across and out the nasal skin incision. Another bite is taken in the tarsus and the suture is then carried up under the orbicularis and tied in the nasal brow incision. The lid is pulled up to a point about two mm. higher than the final desired position before being tied. It is most important that good bites of fascia and tarsus be taken or else the suture will slip.

The cornea is protected for a few days by a modified Frost suture.[25] This suture is passed into the tarsus of the lower lid through the lid margin and the lower lid is pulled up to cover the eye, the suture being taped to the brow. Frost described a suture which went through the margin of both upper and lower lids, under the skin of the upper lid, and was tied above the brow. I have simply passed a similar suture into the tarsus of the lower lid and taped it to the brow. This pulls the lower lid up to cover the

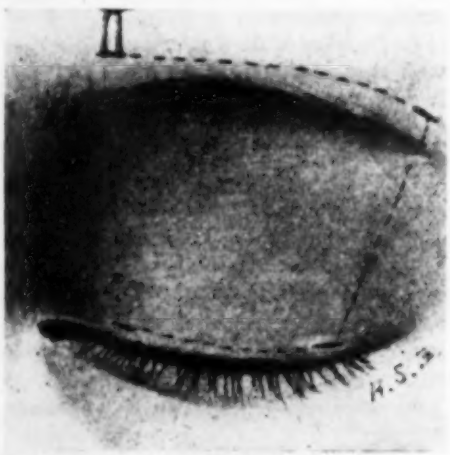


Fig. 5 (Johnson). The *Friedenwald-Guyton* suspension suture, utilizing the frontalis fascia.

TABLE 3

PTOSIS OPERATIONS UTILIZING THE SUPERIOR RECTUS MUSCLE

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Postoperative Complications	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.					
45	6	M	R	8/23/49	Congenital	None	None	3.5	3	0	0	Motais	None	4 mo.	F	Not high enough
			L	8/23/49	Congenital	None	None	4	1.5	0	0	Motais	None	4 mo.	F	Some tenting of lid
49	16	M	R	3/22/50	Congenital	None	Blaskovics	5.5	2.5	0	0	Berke-Motais	None	3 mo.	E	
60	4	M	R	9/25/45	Congenital	None	None	5.5	4	0	0	Dickey	None	7 yr.	G	
			L	9/25/45	Congenital	Convergence	None	7	4	0	0	Dickey	Persistent exposure keratitis	7 yr.	F	Would be considered good except for keratitis
77	5	M	L	2/23/46	Congenital	Convergence	None	6 to 8	5.5	0	0	Dickey	Developed hypotropia	6 yr.	F	Following this operation he had a levator resection by another surgeon without much improvement
	9	M	L	4/11/50	Congenital	Hypotropia	Dickey and lev. resect.	4.5	2	0	0	Berke-Motais and recession of inf. rectus	None	2 yr.	E	
83	50	M	R	1/ 8/46	Congenital	None	None	8 to 9	1	+	+	Dickey	Exposure keratitis; entropion of upper lid	1 yr.	G	Entropion repaired and excess skin resected 2/12/46
			L	1/ 8/46	Congenital	None	None	4	1	++	++	Dickey	None	1 yr.	G	
99	6	M	R	6/21/50	Congenital	Trichiasis due to a recent attempt to correct ptosis	Trainor and some sort of lev. resect.	N.S.	2	0	0	Berke-Motais	Keratitis preop. and postop.	2½ yr.	F	Very distorted lid to work on. Badly mutilated by previous op. Postop. height good, but some exposure keratitis

eye and also splints the upper lid.

There are many obvious advantages to the Friedenwald-Guyton operation. It is simple, only two or three days of hospitalization are required, it may be done to improve a previous unsatisfactory ptosis operation of any type, and it may be repeated easily if necessary.

One disadvantage is that one introduces a foreign body into the tissues of the lid and postoperative infections, both early and late, are more common with this operation than in operations in which no such foreign material is introduced. Friedenwald and Guyton reported three cases with delayed infections around the suture.

In my series, there is one case of cellulitis which came on one week postoperatively (Case 27, Table 4). The cellulitis was easily controlled but the patient continued to have a small draining area in the right upper lid. The suture was removed several months postoperatively. This stopped all evidence of infection and the lid remained in good position.

A second case (Case 67, Table 4) developed a small draining sinus in the region of the suture in her left brow. This came on one year postoperatively, drained a little serum only occasionally, and stopped one year later after the extrusion of a small piece of suture material.

A second complication is that the suture material may break. Friedenwald and Guyton reported the breakage of a 4-0 silk suture when the patient was struck in the brow. In my early cases, I used 4-0 silk and in one case (Case 20, Table 4 this material broke when the patient was struck in the eye. Since then I have used 2-0 braided silk (Deknatel), and, to my knowledge, none have broken. In only one case (Case 21, Table 4) was tantalum wire used.

II. Operations utilizing the superior rectus

This type of operation is indicated chiefly in cases of bilateral ptosis in which the levators are completely paralytic and the superior recti are normal. When the superior rectus is used to suspend the lid some degree of hypotropia is almost sure to result. This is less likely to happen with Berke's modification of the Motais[8] operation than with any other operation of this type, but even with this operation there is apt to be some hypotropia. This is no great problem in a bilateral case, but in a unilateral case with good binocular vision, there is danger of producing diplopia and if this occurs in a young child, suppression and ambloypia may ensue.[31] If binocular vision is absent, this type of operation is excellent in unilateral cases with paralytic levator and good superior rectus.

Another disadvantage is the danger of postoperative keratitis. This is much more frequent, in my experience, in this type than in either of the other two types of operation, and seems to be due chiefly to interference with normal blinking. I have several cases with normal closure in sleep who have persistent exposure keratitis, and the only possible explanation seems to be the lack of blinking. None have any corneal anesthesia (Cases 69, 83, 99, Table 3). The danger of exposure keratitis is greatest in the immediate postoperative period and lessens as time goes on.

The great advantage of this type of operation is that the lid moves synchronously with the eyeball. This is a real advantage over operations in which the lid is suspended from the frontalis.

In this series three different kinds of superior rectus suspension are represented: The Dickey,[2-5] Motais-Parinaud,[6, 7] and Berke's modification of the Motais.[8] Many operations in this category have been devised but one of the simplest and most efficient is the Berke-Motais. In my opinion it is by far the best operation in this category. One great advantage of this operation is that the superior rectus is resected so that there is less hypotropia after this than after any other procedure of this general type. The

TABLE 4

PTOSIS OPERATIONS UTILIZING THE FRONTALIS MUSCLE

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of ptosis		Apparent Levator Action		Type of Operation	Postoperative Complications	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.					
20	8	M	R	8/16/48	Hereditary congenital	Paresis upward gaze and hypertelorism	None	5.5 mm. overlap	3.5 mm. overlap	0	0	F.-G. sling	None	7 mo.	G temporary	Was struck in eye and suture broke, therefore had another operation right eye with excellent result
			L	8/16/48	Hereditary congenital		None	5.5 mm. overlap	3.5 mm. overlap	0	0	F.-G. sling	None	7 mo.	E	
			R	12/20/48	Hereditary congenital		F.-G. sling	4.5	3	0	0	F.-G. sling	None	4 mo.	E	
21	23	M	L	11/ 8/46	III nerve paralysis (congenital)	III nerve paralysis	Fascial sling from frontalis	8	N.S.	0	0	F.-G. sling using tantalum wire	None	2 mo.	F	Only slight improvement
27	16	F	R	11/ 6/52	Congenital	None	None	6.5	5.5 to 2.5	0	0	F.-G. sling	Transient cellulitis	4 mo.	G	
			L	11/ 6/52	Congenital	None	None	7	6 to 3	0	0	F.-G. sling	None	4 mo.	F	
55	16	M	R	12/18/52	Congenital	Epicanthus and blepharophimosis, both eyes	Blair op. for epicanthus, both eyes	7 to 4	5 to 2.5	0	0	F.-G. sling	None	4 mo.	E	
			L	12/18/52	Congenital			7 to 4	5 to 2.5	0	0	F.-G. sling	Corneal abrasion from pressure bandage	4 mo.	E	
56	16	F	L	2/21/49	→	Complete paralysis left III nerve—dilated pupil; hypoexotropia	?Lev. resect.	8 to 10	5.5 to 3.5	0	0	F.-G. sling	Slight but persistent exposure keratitis	4 yr.	F	Can raise lid to within 1 mm. of height of O.D. by raising brow, but seldom does so
67	51	F	R	8/17/50	Prog. ophthalmoplegia ex. terna O.U.	All E.O.M. paretic	1932 had Reese ptosis op. both eyes	6.5	4.5	0	0	F.-G. sling	None	3 yr.	G	One-year postop. overlapped cornea 0 to 3 mm.
			L	8/17/50		All E.O.M. paretic		6.5	3.5	0	0	F.-G. sling	Delayed low-grade infection left brow	3 yr.	G	One-year postop. overlapped cornea 0 to 3 mm.
78	22	M	R	9/28/50	Congenital	Blepharophimosis and oxycephaly	None	5.5	3.5	0	0	F.-G. sling & canthoplasty	Non-	3 mo.	G	
			L	9/28/50	Congenital	Blepharophimosis and oxycephaly	None	5.5	3.5	0	0	F.-G. sling & canthoplasty	Closure of canthoplasty	3 mo.	G	Reoperation on external canthus 11/9/50 with good result
84	5	M	R	10/16/52	Congenital	Blepharophimosis and oxycephaly	None	5.5 to 4.5	2.5 to 0	0	0	F.-G. sling	None	3 mo.	E	
			L	10/16/52	Congenital	Blepharophimosis and oxycephaly	None	5 to 4	3 to 0.5	0	0	F.-G. sling	None	3 mo.	E	

following description is mine, rather than Berke's, and contains a few very minor modifications of his technique:

An incision is made in the skin of the lid about five or six mm. above the lid margin (fig. 6). The skin and orbicularis are then freed up nearly to the lash line. An incision is made through levator and conjunctiva just at the upper tarsal border. Next an incision is made as shown in Figure 7 over the superior rectus insertion. The muscle is divided into three equal bundles and a suture is tied tightly around each about two mm. from its insertion as shown in Figure 9, and the insertion is cut. Berke uses catgut but I prefer 5-0 braided white silk.

The muscle is reattached and the conjunctiva closed by means of the two 4-0, chromic catgut sutures shown in Figures 10 and 11. The amount of shortening varies. If there is no demonstrable weakness of the superior rectus, about five to seven mm. should be about right. If there is weakness, up to 10 mm. may be taken and one can also recess the inferior rectus at the same time. I then pass the three sutures which are tied around the muscle bundles, into the *tarsus*, regulating the height of the lid to approximate that of the other eye. One should overcorrect one to two mm.

Figure 13 shows Berke's method of passing the sutures out the lid margin. I prefer to bury the silk in muscle and tarsus.

If there is excess skin, it now becomes evident and may be excised.

The skin is closed with interrupted silk sutures in a manner which I shall describe later in speaking of levator resection.

A modified Frost suture[25] is next placed in the lower lid margin; that is, into the intermarginal strip and deep into the tarsal plate. This suture is fastened to the brow, pulling the lower lid up to cover the eye (see above).

The operation is completed by a pressure bandage which is left in place for three days. The skin sutures may be left in place for five days.

III. Levator resection and/or advancement

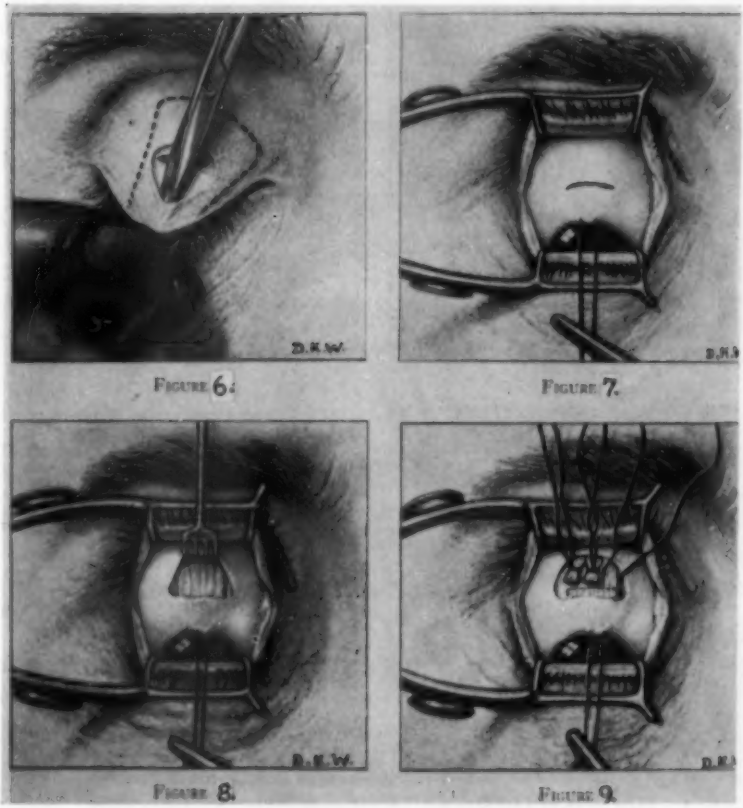
This type of procedure has by far the widest application. Blaskovicz[1] said that his operation, which is probably the best known operation in this category was applicable to any type of ptosis. This is probably true, but in some instances a suspension from frontalis or superior rectus may give a more pleasing result (see above).

Of course, such a procedure cannot be used if there is no levator muscle. However, cases in which there is neither muscle, aponeurosis, nor fibrous tissue in place of muscle must be extremely rare. Such a condition is mentioned in the literature, but I have not yet encountered it.

Advantages. Among the advantages of levator resection are:

1. Postoperative reaction is negligible.
2. Lagophthalmos is rare.
3. If there is any levator function, the lid moves with the globe.
4. The lid margin is normal. There should be no tenting of the margin if the operation is performed properly.
5. There is no danger of postoperative diplopia as there is in unilateral cases when the superior rectus is utilized to suspend the lid.
6. *The winking reflex is not disturbed.* This is an important point. Whenever the lid is attached to brow or superior rectus there is more or less interference with the normal winking reflex, whereas this reflex is not interfered with when the levator is shortened. Many cases of exposure keratitis following the first two types of operation seem to be due entirely to this factor. The patients close their eyes well enough in sleep, but wink incompletely and infrequently during the day. This is, of course, more noticeable when the superior rectus is used to suspend the lid than when the frontalis is used.

Disadvantages. Theoretically, it should not be effective if there is no levator action. Actually it will be, but perhaps not quite so effective as one of the other types in such



Figs. 6 to 9 (Johnson). *Modification of Berke's ptosis operation.*

(Fig. 6) Incision 20 to 25 mm. long through skin and orbicularis. The incision through levator and conjunctiva at upper tarsal border is seen behind the scissors.

(Fig. 7) Incision through conjunctiva over the superior rectus insertion. The suture is for traction.

(Fig. 8) Exposure and freeing of superior rectus.

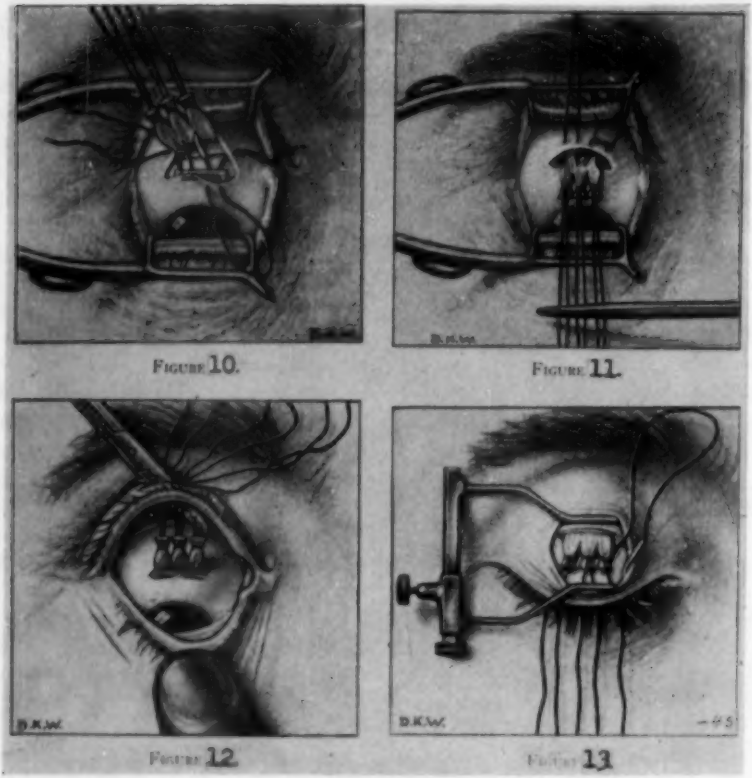
(Fig. 9) Placing of sutures around three bundles of superior rectus tendon 2-3 mm. from its insertion. The author prefers 5-0 double-armed white silk sutures.

cases. However, from a cosmetic standpoint, I prefer it to suspension from the frontalis in most unilateral cases with an accompanying weak superior rectus unless the patient spontaneously uses his brow to lift his lid before operation. In unilateral cases with good superior rectus function, I prefer it to suspension from the superior rectus if there is binocular vision.

Resection of the levator is, in my opinion, the operation of choice in most cases in which there is any levator action whatsoever, even if that action be indicated simply by a deepening of the lid fold in upward gaze. When the operation which I shall describe is done, many cases which show only this much evidence of function preoperatively have good lid motion postoperatively.

This point is worth emphasis because many surgeons consider that levator resection is indicated only when good levator function can be demonstrated preoperatively.

Levator resection also produces a satisfactory result in many cases of unilateral third



Figs. 10 to 13 (Johnson). *Modification of Berke's ptosis operation.*

(Fig. 10) Re-attaching and shortening the superior rectus. The figure shows Berke's ingenious method of closing conjunctiva, and shortening the superior rectus using two double-armed, 4-0 chromic catgut sutures. The sutures are passed first through the lower lip of the conjunctiva, then through the muscle stump, then through the superior rectus, and finally through the upper lip of conjunctiva and tied.

(Fig. 11) The sutures tied, leaving the three tongues of superior rectus exposed.

(Fig. 12) Pulling the three tongues of superior rectus through the incision in the lid.

(Fig. 13) The three tongues of superior rectus muscle spread out on the tarsus. The distance between sets of sutures should be about five mm., and the author prefers to suture the muscle to the anterior tarsal surface instead of passing them out the lid margin as shown here.

nerve paralysis and congenital paralysis of upward gaze (see Cases 2, 41, 89, 94, 97, Table 2). Suspension from the frontalis would seem to be the procedure of choice in such cases, but the patient is more likely to have exposure keratitis with this than with levator resection, due to interference with the winking reflex. In addition, as has been pointed out previously, suspension from the frontalis is less often satisfactory in unilateral than in bilateral ptosis.

I prefer levator resection also in many ptosis cases with the Marcus Gunn phenomenon. In many instances the winking is not too noticeable and levator resection produces a good functional result. Sometimes, however, the winking is the most prominent feature. In such cases the levator should be sectioned and the lid suspended from the frontalis, as advocated by Spaeth,[20] or, if fusion is weak and the superior rectus reasonably strong, the Berke-Motais opera-

tion is the operation to be preferred.

Traumatic cases are occasionally seen in which no levator function can be demonstrated preoperatively. If the trauma was in an accessible portion of the lid, or orbit, it is worth while to try to find the proximal portion of the levator and reattach it to the tarsus. This is much easier than might be expected, especially if the operation is done under local anesthesia so that one can look for movement of the muscle when the patient is directed to look up and down (see Cases 44, 64, 80, Table 1).

Eversbusch's name is frequently given to various levator resection operations which are done through the skin surface. Actually the operation he described was a tucking of the levator.[16] The levator resection described in this article was based on de Lapersonne's operation[17] but has been somewhat modified. I have been using the operation since 1941.

The Blaskovicz operation[1] and its modifications[2, 18] are very satisfactory procedures, but the levator can be freed up more easily from the anterior surface, exposure is better, and a larger resection can be done. In doing Blaskovicz's operation, one operates with the lid everted, and mechanically it is difficult, in many instances, to do a large enough resection of the levator. It is also more difficult to get a good, firm reattachment of levator to tarsus.

Blaskovicz[1] simply passed the sutures out through the skin in such a way that the levator was approximated to the resected tarsus, but not actually sutured to it. This allows for slippage and undercorrection. It is very nearly impossible to overcorrect a ptosis by the Blascovicz method, so in doing the operation one usually tries to resect as much of the levator as is possible. This is not true when the operation is done from the anterior surface, so it is easier to judge the proper amount of levator to be resected.

Levator resection

The operation may be done under either local or general anesthesia. If local anesthesia is used, a retrobulbar injection of four-percent procaine with 1:50,000 epinephrine is first given, then both lids are infiltrated with the same solution.

I perform the operation as follows:

An incision is made the full length of the upper lid about six mm. above the margin (fig. 14). (This is the usual location of the upper lid fold.) The incision goes through skin and orbicularis to the tarsus.

It is important to make the incision on the tarsus but the exact height can be varied slightly in order to match the other lid (in a unilateral case). If the incision is made higher up, the anatomy is more confusing and it is more difficult to make a new lid fold.

Dissection is next carried down close to the lid margin and upward as high as possible, keeping close to the levator aponeurosis and beneath the septum orbitale (fig. 15).

A buttonhole incision is made through levator and conjunctiva at the upper border of the tarsus near its inner end. A similar incision is made near the outer end and a hemostat or Berke's clamp[18] is placed on the combined levator aponeurosis, Mueller's muscle, and conjunctiva (fig. 16) which are then cut off at the upper border of the tarsus.

No tarsus is resected. Resection of the tarsus is not only unnecessary, but is to be avoided. If tarsus is resected, there is more likely to be tenting of the lid margin and it is also more difficult to keep the lashes in proper position.

The clamp is rolled upward and the conjunctiva is then easily dissected off Mueller's muscle (fig. 17). The levator and Mueller's muscle may be resected without cutting the conjunctiva, but it is somewhat difficult to do this without shredding the thin aponeurosis. The conjunctiva can then be loosely sutured to the upper tarsal margin with a running silk suture, the ends of which are carried out through the skin.

Freeing up of the central two thirds of the muscle is completed by cutting the tendonous medial and lateral horns. This must be care-

Fig. 14 (Johnson). Lid incision for levator resection. An entropion clamp is used at this stage to keep the operative field bloodless.

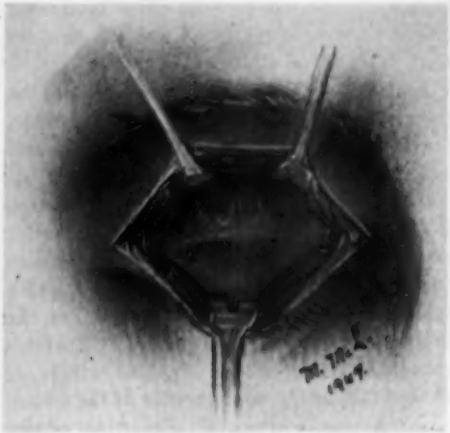


Fig. 15 (Johnson). Exposure of tarsus and levator aponeurosis.

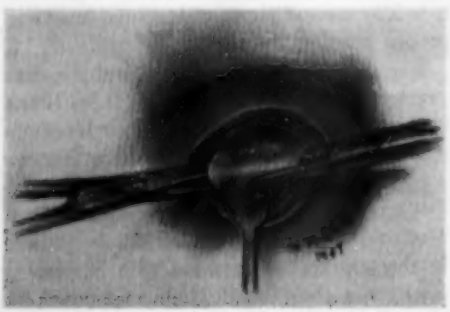


Fig. 16 (Johnson). Placing of hemostat (or Berke's ptosis clamp). The clamp is placed just above the upper border of the tarsus. In its grasp it has levator aponeurosis, Mueller's muscle, and conjunctiva.

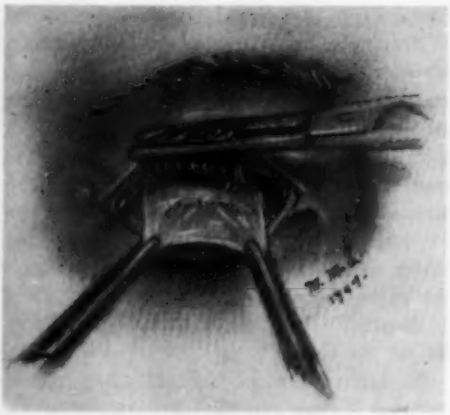


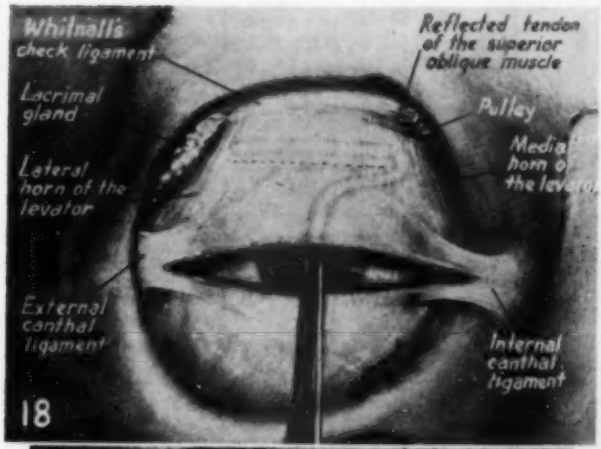
Fig. 17 (Johnson). Freeing conjunctiva from Mueller's muscle which underlies the levator aponeurosis. The aponeurosis and Mueller's muscle are still in the grasp of the hemostat but the conjunctiva has been freed upward to the fornix.

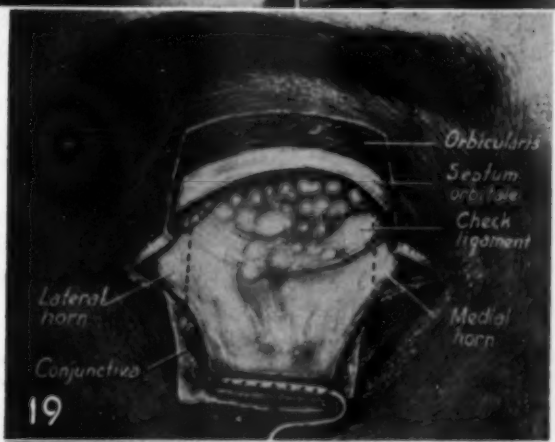
fully done in order not to damage the underlying superior rectus and superior oblique. One must also be thorough or the levator will be insufficiently freed up. This will result in an unnecessary restriction of movement in downward gaze. The following three figures from Berke[2] (figs. 18, 19, and 20) illustrate the anatomy involved and the dotted lines in Figure 19 illustrate the points to be incised.

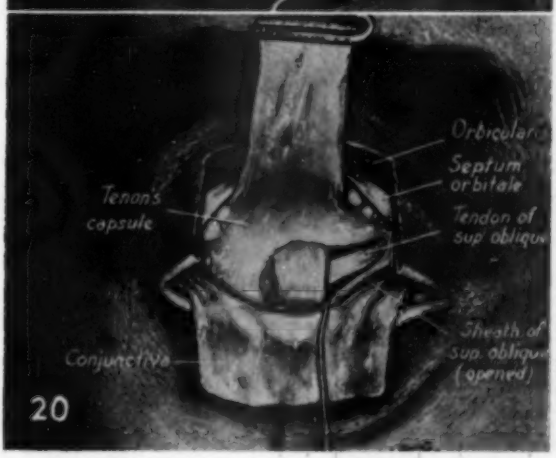
The lid is then pulled up to the desired position in order to judge where to place the sutures. If there is no levator action or if it is minimal, the lid should be overcorrected one mm. or so. If there is good levator action, it is best to undercorrect one mm. On the average, about 15 mm. of levator aponeurosis is resected (fig. 21), but the amount resected may vary from five to 25 mm. depending upon the amount of ptosis and the elasticity of the levator.

Three or four 5-0 braided white silk sutures are placed in a straight line in the anterior surface of the aponeurosis or muscle and the excess muscle is excised. Each of the needles is then passed into the anterior surface of the tarsus about two mm. above the lashes (fig. 22).

The bite in each case is horizontal and if







Figs. 18, 19, and 20 (Johnson). *Berke's ptosis operation.*

(Fig. 18). *Insertions of the levator aponeurosis.* Phantom view of the position of the ptosis clamp preparatory to freeing the aponeurosis in resection of the levator muscle. Note the close relationship between the medial third of Whitnall's check ligament and the reflected tendon of the superior oblique muscle. Dotted line indicates site of incision for severing the aponeurosis from the tarsus. (after Berke).

(Fig. 19). *Schematic drawing illustrating the position of the levator after it has been freed from the tarsus and pulled downward.* Part of the upper lid has been excised to show the deeper structures. Note that the lateral horn, the medial horn, and the check ligament restrict pulling of the levator downward. The dotted lines indicate the site of incision of these structures in freeing the levator muscle during resection of the levator (after Berke).

(Fig. 20). *Schematic drawing illustrating the relationship of the levator muscle (after it has been freed) to Tenon's capsule, to the superior rectus muscle, and to the reflected tendon of the superior oblique muscle.* Tenon's capsule has been opened and part of the upper lid excised to demonstrate these points (after Berke).

TABLE 5

COMBINATIONS OF TWO OR MORE TYPES OF OPERATION
(Included here are canthoplasties done for ptosis)

Case No.	Age	Sex	Eye	Date of Operation	Cause	Associated Anomalies	Previous Operations	Amount of Ptosis		Apparent Levator Action		Type of Operation	Amount Resected	Postoperative Complications	Length of Follow-up	Result	Remarks
								Preop.	Postop.	Preop.	Postop.						
13	5½	F	R	10/12/49	Congenital	Blepharophimosis and epicanthus	None	N.S.	Both lids same height	+++	+++	Canthoplasty		None	2 yr.	G	Lids almost exactly same height
			L	10/12/49	Congenital		None	2 mm. lower than O.D.		+++	+++	Canthoplasty		None	2 yr.	G	
25	44	M	R	2/23/45	Congenital	Blepharophimosis	None	6	5	++	++	Canthoplasty		None	8 yr.	F	
			L	2/23/45	Congenital	Blepharophimosis	None	5.5	5	++	++	Canthoplasty		None	8 yr.	F	
	46	M	R	2/27/50	Congenital	None	Canthoplasty	5	0 to +1	++	++	Lev. resect.	14	Several chalazia	3 yr.	E	
			L	2/27/50	Congenital	None	Canthoplasty	5	0 to +1	++	++	Lev. resect.	14	None	3 yr.	E	
26	3	M	L	8/23/47	Congenital	None	None	5	5	0	0	Dickey		Stitch abscess in brow	3 yr.	U	
	4		L	8/15/48	Congenital	None	Dickey	5	5	0	0	F.-G. sling		Suture slipped or broke	2 yr.	U	
	4		L	10/18/48	Congenital	None	Dickey and F.-G. sling	5	5	0	0	Lev. tucking	3 mm.	None	2 yr.	U	
54	4	M	R	5/10/51	Congenital	Epicanthus and Blepharophimosis both eyes	Repair of epicanthus both eyes	5.5	5.5	0	0	Lev. resect.	7 mm.	None	2 yr.	U	
	4		L	5/10/51	Congenital			4.5	3	+	+++	Lev. resect.	7 mm.	None	2 yr.	G	
	4		R	8/23/51	Congenital	Epicanthus and blepharophimosis both eyes	Lev. resect.	5.5	6	0	0	F.-G. sling		None	1½ yr.	U	Lid was elevated so it overlapped 4 mm., until struck on brow 9/26/51; it then dropped to 5.5 to 6 mm.
	5		R	9/25/52	Congenital	Epicanthus and blepharophimosis both eyes	Lev. resect. and F.-G. sling	6	4.5 (temporary)	0	+	Lev. resect.	20 mm.	Ectropion outer half, upper lid	6 mo.	F	
59	21	M	R	5/31/45	Congenital	None	None	5	5	0	0	Lev. resect.	10 mm.	None	7 mo.	U	
				12/17/45	Congenital	None	Lev. resect.	5	2	0	0	Dickey		None	4 mo.	E	4Δ hypotropia, but no symptoms and looks excellent

the needle went from right to left in the muscle, it goes from left to right in the tarsus. This gives the effect of a mattress suture with only one needle.

By placing the sutures in this fashion, there is no danger of catching the conjunctiva between muscle and tarsus, as the muscle rolls over when the suture is tied, so that a little of its previously anterior surface is in contact with the tarsus. In addition, there is less chance for the muscle fibers to slip out of the tied suture.

The sutures are next tied once and if the curve of the lid is not proper, or if it is too high, it is a simple matter to pull the needle back out of the tarsus and replace it higher up. If there is excess skin, it may now be excised.

It should be noted that this procedure is an advancement as well as a resection of the levator. Because of the method of advancement it also lends itself quite easily to a recession in cases of overcorrection. This was necessary in two cases in this series (Cases 8 and 29, Table 1). The levator can easily be recessed to the upper tarsal border.

The skin is now sutured, and this suturing is very important.[23] If not properly done, there will not be a good skin fold and there may be trichiasis or at least an unnatural position of the lashes, which will greatly detract from the final appearance of the lid.

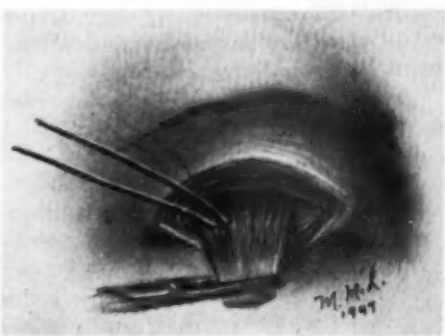


Fig. 21 (Johnson). Method of placing sutures in levator. Each suture takes a two- or three-mm. bite and three or four single-armed 5-0 white silk sutures are used.

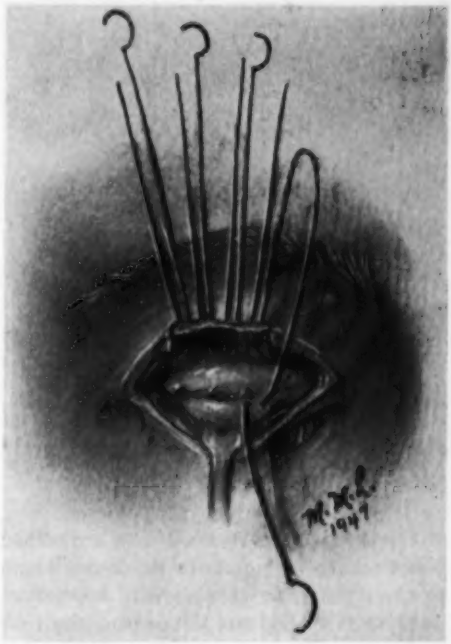


Fig. 22 (Johnson). Method of placing sutures in tarsus. It is to be noted that each suture goes into the anterior surface of the levator from right to left and into the tarsus near its lower border from left to right. This produces a mattress suture although only one needle is used; it also causes the end of the muscle to roll under, thus lessening the possibility of slippage of the sutures. The conjunctiva can be seen loosely sutured to the upper tarsal border.

Usually three or four 4-0 silk sutures are used to produce the desired result (fig. 23). Each suture goes through the lower skin edge, then through the levator and into the tarsus high enough so that the lower skin flap is put on a slight stretch, then out through the upper skin edge. The sutures are tied just tightly enough to make a new skin fold of moderate depth. Any additional closure of the skin which may be needed can be obtained by means of ordinary interrupted skin sutures. This method of suturing holds the skin tightly against the tarsus, makes a new lid fold, and holds the lashes up in their proper position. In addition, it helps to reapproximate levator fibers to skin as well as to tarsus.

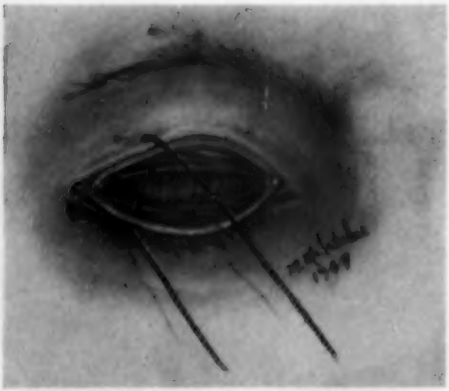


Fig. 23 (Johnson). Closure of the operative incision. The skin sutures are passed through the advanced levator and into the tarsus. This produces a new lid fold and brings the lashes up into proper position.

The operation is finished with a modified Frost suture[25] which pulls the lower lid up to cover the globe (see above). A pressure dressing is applied and left in place for four to five days. The sutures are usually removed and the patient discharged on the fifth postoperative day. All ptosis patients are given methyl-cellulose drops or boric ointment to use three times a day until all danger of exposure keratitis is passed.

It has been stated[2] that in the Blaskovicz operation overcorrection at the time of the first dressing is important. If it is not overcorrected at that time, the ultimate result will be an undercorrection.

In the levator resection just described, this is also true if there is no levator action or if the action is minimal, but it is definitely not true in cases with good levator action. In cases with poor levator function, I like to see a one-mm. overcorrection at the time of the first dressing but a one-mm. undercorrection in cases with good levator function.

In the latter type of case, in my series, the lids have tended to rise higher in the first week or so after the dressing has been removed. I have ascribed this to the effect of the pressure dressing—the pressure causing a temporary paresis of the levator which regains function and raises the lid further after the pressure has been removed.

In cases done with procaine anesthesia, the procaine also contributes to this temporary paresis. This effect is obviously of very little importance when the levator is nonfunctioning.

Levator tucking

There are some cases of ptosis in which the ptosis is only moderate and in which the levator action is nearly normal. These cases may be cured by a simple tucking[10, 24] of the levator without an extensive levator resection (see Case 113, Table 1). Cases 2 and 61, Table 2).

An incision is made as for levator resection and the anterior surface of the aponeurosis is exposed. This exposure need not be carried up more than 10 mm. above the tarsus. Three small bundles of levator aponeurosis are partially freed up from the underlying conjunctiva, sutures are placed as for levator resection, but the bundles are simply brought forward and fastened to the anterior tarsal surface as in levator resection. The skin is then closed in the usual manner (see above).

The following outline is a resume of the indications for the three types of operation described. This is simply a guide, as no absolute rules can be laid down.

I. Unilateral
   - A. With some, even very slight, levator action—levator resection
   - B. With no levator action
     1. With fair or good superior rectus function
        - a. If strong binocular vision—levator resection
        - b. If no binocular vision or if patient suppresses easily—Berke-Motais operation
     2. With no or poor superior rectus function—variable, usually levator resection, but frontalis suspension if patient spontaneously uses brow to help elevate lid.

II. Bilateral

A. With some, even very slight, levator action—levator resection

B. With no levator action

1. With no or poor superior rectus function—frontalis suspension

2. With fair or good superior rectus function—first choice, Berke-Motais operation; second choice, frontalis suspension

Explanation of the tables

I have divided my cases into five groups, as shown in Tables 1 to 5:

I. *Levator resections* in cases uncomplicated by weakness of elevation or weakness of the superior rectus alone. Included in this group are traumatic cases and cases with blepharochalasis and with acquired blepharophimosis. None of my patients with congenital blepharophimosis has had normal superior recti.

II. *Levator resections* in eyes with weak or paralytic superior recti, with weakness of upward gaze, with the jaw-winking phenomenon, or with third-nerve paralyses. In this group are also those cases of congenital blepharophimosis and epicanthus in which levator resections were done as the only ptosis procedure—usually with or following a canthoplasty, and, in some cases, repair of the epicanthus. (As already noted none of these had normal superior recti.)

III. *This group* includes three different operations—the Motais, the Dickey, and the Berke modification of the Motais-Parinaud operation.

IV. *All in this group* are Friedenwald-Guyton sling sutures. In the early cases, twisted white silk was used except in one case, which was repaired with tantalum wire. For the past several years, however, I have used 2-0 braided silk (Deknatel).

V. *This is a mixed group* of cases in which more than one type of ptosis procedure was used. For want of a better place I have included here four eyes in which canthoplasties were done as a ptosis operation, rather than combined with a ptosis operation as they were in many cases in the previous categories.

In one of these cases (Case 13, Table 5—two eyes), the canthoplasty alone was all that was ever necessary to give a good result. In the second case (Case 25, Table 5)—two eyes), the height of the lids was improved so much by canthoplasty alone that the patient waited five years before deciding to have more done.

The remaining cases in this group are obviously difficult ones, which helps explain the combinations of operations.

In the tables the "amount of ptosis" is usually listed as the amount of corneal overlap in the primary position with the brow held (see above). In most cases of the Marcus Gunn phenomenon the maximum height (jaw opened and pushed to side opposite the ptosis), and minimum height (jaw closed) are given. Minus (−) before a figure refers to height *above* the limbus. A figure alone signifies the amount of corneal overlap in millimeters. When an exact figure could not be found in the patient's record, the abbreviation N.S. (not stated), is used.

Length of follow-up refers not to the length of time which has elapsed since the operation was done, but to the length of time from operation until the patient was last seen.

Results

The accompanying tables show the results of surgery in the cases reported. In grading results, the following were the criteria used:

Unimproved (U)

A lid was called unimproved if not *appreciably* better than before operation. All poor results are included here.

Fair (F)

A result was called fair if there was an *appreciable* improvement over the preoperative appearance, with good contour of the lid margin, even if it were not as high as the

fellow eye. In a case in which I believe that the best possible result for that particular case had been achieved, I still did not list the result as good or excellent unless it met the criteria for good or excellent as given below.

Good (G)

A result was called good in a unilateral case if there was 1.5 mm. or less difference in the height of the two lids and the result was pleasing in appearance, again without any unevenness of the lid margin. In a bilateral case, obviously the result on one lid might be called good, while the result on the other lid might be fair or unimproved.

Excellent (E)

A result was called excellent in a unilateral case if the operated lid was within 0.5 mm. of the height of the fellow eye, and if there was no great difference in their height in looking up and down. No matter what the height of the center of the lid, a case was not called excellent if there was any unevenness or any semblance of tenting of the center of the lid. In a bilateral case the result was called excellent if the two lids overlapped the corneas 2.5 mm. or less in the primary position, and functioned the same in looking up and down. Fortunately no cases in this series were made worse by operation.

These criteria are admittedly unsatisfactory because a pleasing cosmetic result cannot be judged on figures alone. The height of the lid in the primary position is not all important. If the patient's lids are the same height in the primary position but there is an unsightly lag in looking down, the result cannot be considered good or excellent —such a patient would look better a greater share of the time had the final height of his lid in the primary position been a little lower than the fellow eye.

As an example, consider Case 97, Table 2, figure 33. This child has a paralysis of the elevators of her left eye, and before ptosis surgery she had extraocular muscle surgery to bring the eye in and up. A levator resection was done on the paralyzed muscle. The end result was an undercorrection of 1.5 mm., yet the appearance is pleasing, the patient is happy, and she has had no trouble with her cornea. I feel quite sure that had the lid been raised 1.5 mm. higher she would have had exposure keratitis. She is listed in the table as a good result although for such a case it might be considered excellent.

It has been my experience that patients are pleased with an undercorrection more often than they are with an overcorrection and I also believe that they look better. Even a slight overcorrection is apt to produce a staring expression. Furthermore, an overcorrection is much more likely to produce exposure keratitis—especially in operations in which the superior rectus is utilized.

Operative results

I. Good and excellent on basis all operations = 67 percent of 162 operations.

II. Good and excellent on basis all eyes = 76 percent of 142 eyes.

1. Levator resection with good superior rectus = 85 percent of 78 eyes.
2. Levator resection with poor superior rectus and/or congenital blepharophimosis and/or epicanthus = 56 percent of 39 eyes.

III. Total operations unimproved = nine percent of 162 operations.

IV. Total eyes unimproved = five percent of 142 eyes.

If the correction of ptosis were a wholly satisfactory procedure, there would not be so very many different types of operation. In some instances a poor result is obtained because one has very little to work with; more often, however, it is because one chooses the wrong procedure or uses the wrong technique in doing what would otherwise be the proper procedure.

Obviously it is unwise to advocate any one type of operation for all types of ptosis.

If the results of operations in all cases

in this series are combined, including the extremely unfavorable ones, as well as the favorable ones and including all three general types of ptosis operations done, there are 67 percent in the combined excellent and good group—hardly a brilliant result. But if eyes are used instead of operations as a basis for the figures, there are now 76 percent in the excellent and good group. In other words, a second operation in many cases converts an unimproved or fair result into an excellent or good result.

If the figures are narrowed still further, and we consider only *levator resections* done in eyes with a normally functioning superior rectus, leaving out those few eyes with epicanthus and *congenital* blepharophimosis, the combined excellent and good result jumps to 85 percent.

Included here are all cases from Table 1, and Case 59 from Table 5. Some of these cases (Cases 4, 18, 38, 44, 68, 75, 80, 100, 108, 112) had had previous ptosis operations, some were traumatic (Cases 17, 44, 64, 80, 95, 103), some were accompanied by acquired blepharophimosis (Cases 95, 113), but none had congenital blepharophimosis. Eleven eyes had no levator function.

On the other hand, if we consider only levator resections done in what might be considered complicated cases (all in Table 2 and all levator resections except Case 59 in Table 5)—cases with weak or paralytic superior recti and/or congenital blepharophimosis and/or epicanthus, and/or jaw-winking, the percentage of good and excellent results on the basis of eyes operated upon falls to 56 percent, the lowest of all.

There are not enough frontalis or superior rectus operations in this series to warrant separate statistical analysis.

Fig. 24 (Johnson). Case 16, Table 1. Excellent result. (A) Preoperative. (B) Postoperative.

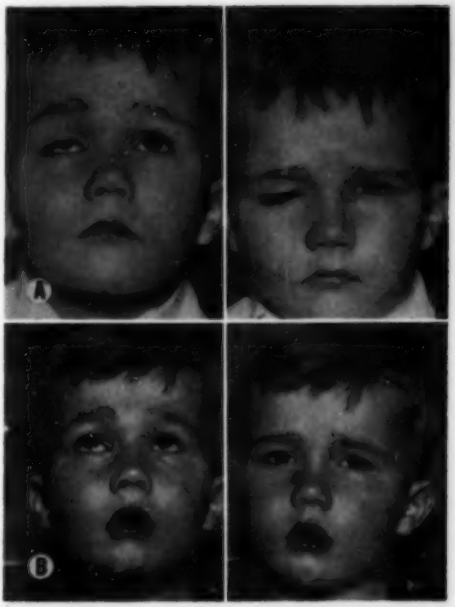


Fig. 25 (Johnson). Case 17, Table 1. Excellent result. (A) Preoperative. (B) Postoperative.

Photographic illustration of results

The following are some cases which illustrate the results previously discussed.

I. Excellent results

In the first group are the following (figs. 24 to 30).

Case 16 (table 1)

Congenital ptosis, listed as excellent result. +++ levator action preoperatively and ++++ postoperatively. Operation was a levator resection, O.D. The postoperative pictures were taken 19 months after operation (fig. 24).

Case 17 (table 1)

Listed as excellent. Ptosis secondary to removal of orbital dermoid. Operation was levator resection, O.D. Postoperative pictures were taken three months after operation (fig. 25).

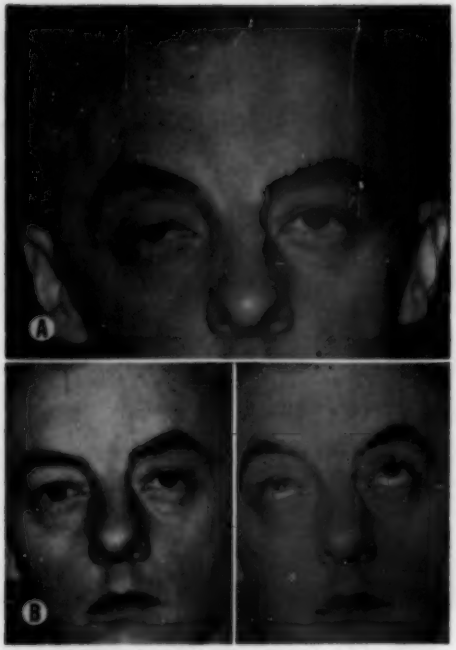


Fig. 26 (Johnson). Case 74, Table 1. Excellent result. (A) Preoperative. (B) Postoperative.

CASE 74 (table 1)

Listed as an excellent result of a levator resection in a case of congenital ptosis of the right upper lid. Postoperative picture taken only two weeks after operation. Patient was followed for one year and height of lid did not change (fig. 26).

CASE 87 (table 1)

Listed as an excellent result of a levator resection in a case of congenital ptosis. Postoperative picture taken three weeks after operation (fig. 27).

CASE 96 (table 1)

Bilateral congenital ptosis plus blepharochalasis. The only evidence of levator action preoperatively was the presence of a lid fold. After bilateral levator resection and resection of excess skin, he showed an apparent +++ action of both levators. Listed as excellent (fig. 28).

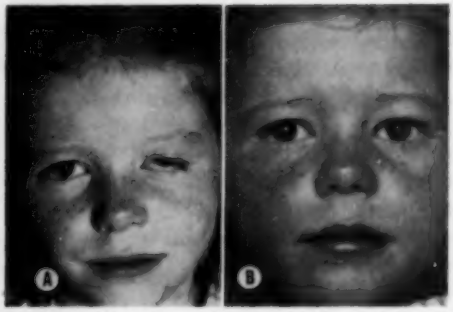


Fig. 27 (Johnson). Case 87, Table 1. Excellent result. (A) Preoperative. (B) Postoperative.

CASE 77 (table 3)

Listed as an excellent result of Berke's modification of the Motais operation. This boy had previously had a Dickey operation with development of a hypotropia. At operation the inferior rectus was recessed. Resection of the superior rectus is part of the Berke-Motais procedure. Postoperative picture taken two months after operation. He was followed for two years after this and did not have any recurrence of ptosis or hypotropia (fig. 29).

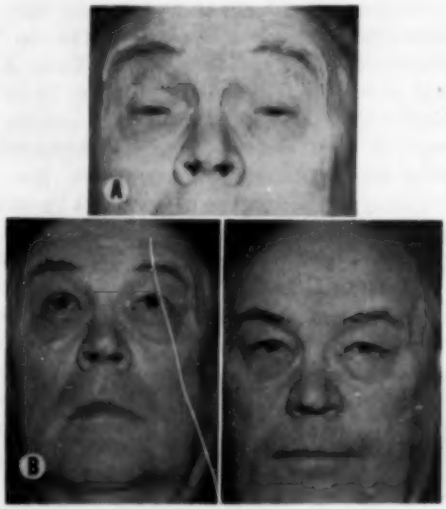


Fig. 28 (Johnson). Case 96, Table 1. Excellent result. (A) Preoperative. (B) Postoperative.

CASE 55 (table 4)

Listed as an excellent result of a bilateral Friedenwald-Guyton frontalis sling suture. The first picture shows him before any operation. In addition to ptosis, there is bilateral inverse type epicanthus and blepharophimosis. The second picture was taken six years later. Marked keloid formation followed a Blair operation for epicanthus but had entirely cleared in the second picture. The third picture shows the result one month after ptosis surgery (fig. 30).

II. GOOD RESULTS

The second group is composed of some examples of those listed as good results (figs. 31 to 33).

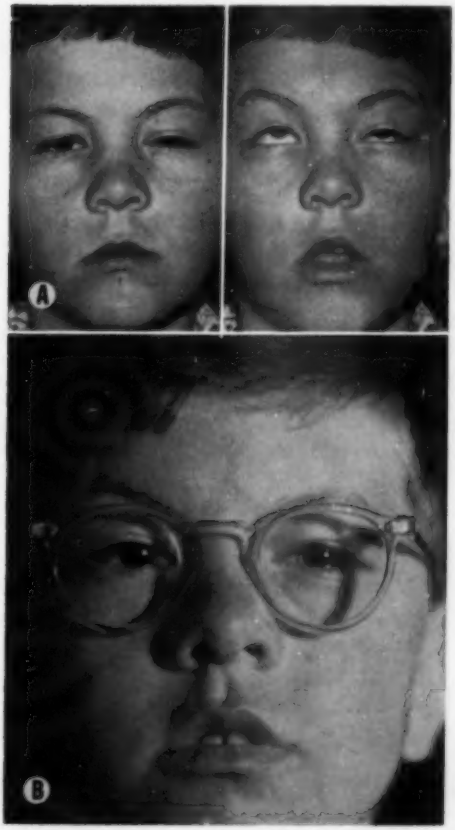


Fig. 29 (Johnson). Case 77, Table 3. Excellent result. (A) Preoperative. (B) Postoperative.

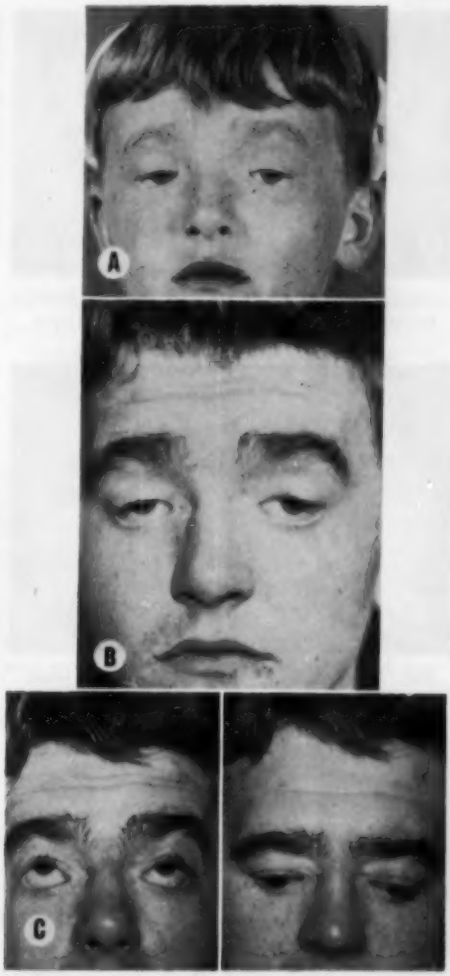


Fig. 30 (Johnson). Case 55, Table 4. Excellent result. (A) Preoperative. (B) Six years later. (C) One month after ptosis surgery.

CASE 71 (table 1)

This child actually had ptosis of both lids, but only the right was operated upon. Listed as a good result of a levator resection (fig. 31).

CASE 73 (table 1)

Listed as a good result of levator resection

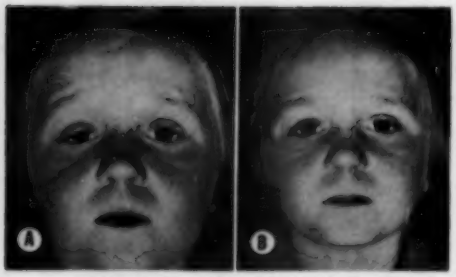


Fig. 31 (Johnson). Case 71, Table 1. Good result. (A) Preoperative. (B) Postoperative.

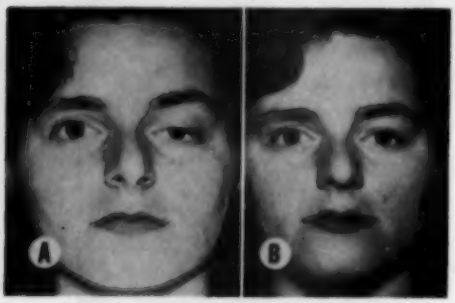


Fig. 32 (Johnson). Case 73, Table 1. Good result. (A) Preoperative. (B) Postoperative.

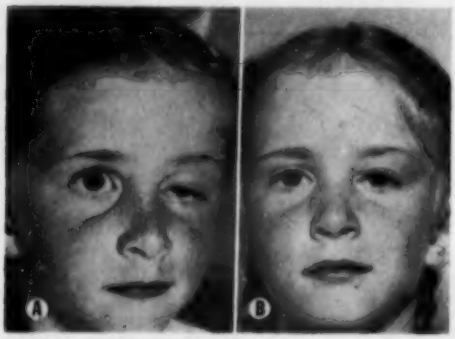


Fig. 33 (Johnson). Case 97, Table 2. Good result. (A) Preoperative. (B) Postoperative.

in a case of congenital ptosis with only + levator action (fig. 32).

CASE 97 (table 2)

This child had a paresis of upward gaze and ptosis with no levator action. After an operation was done for the hypotropia, she had a levator resection. For such a case this might be considered an excellent result. It is listed as good.

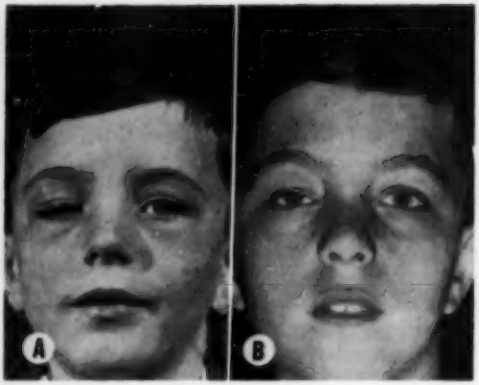


Fig. 34 (Johnson). Case 7, Table 1. Fair result. (A) Preoperative. (B) Postoperative.

III. FAIR RESULTS

The third group comprises some examples of cases listed as only fair results (figs. 34 to 36).

CASE 7 (table 1)

Listed as a fair result of levator resection in a case of unilateral ptosis with no levator action. A lid fold is evident in the preoperative picture but motion of the levator was less than two mm. (fig. 34).

CASE 52 (table 1)

Another example of a fair result of levator

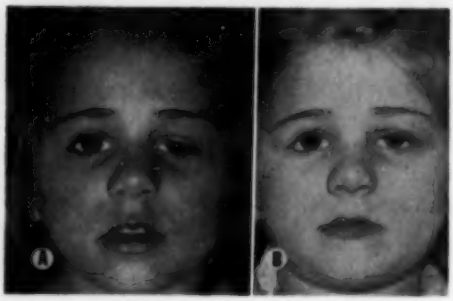


Fig. 35 (Johnson). Case 52, Table 1. Fair result. (A) Preoperative. (B) Postoperative.

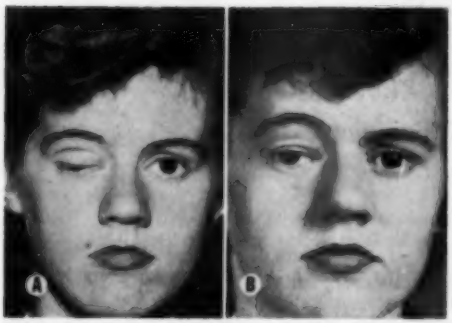


Fig. 36 (Johnson). Case 70, Table 2. Fair result. (A) Preoperative. (B) Postoperative.

resection in a case of unilateral congenital ptosis (fig. 35).

CASE 70 (table 2)

This patient had previously been operated upon at another hospital for removal of an orbital tumor. She had ptosis and paresis of superior oblique, medial rectus, and inferior rectus. A levator resection was done and at operation only the outer one third of the levator could be found. This was resected and the result is considered fair. The postoperative picture was taken two months after operation. One and one-half years later, the lid was in the same position (fig. 36).

SUMMARY

The preoperative investigation of ptosis cases is discussed. It is usually advisable to correct other lid and extraocular muscle anomalies before correcting the ptosis. It is most important to know whether or not the superior rectus functions properly because the results of levator resections are poorer in cases with weak superior recti than in cases with good superior recti. In addition, it usually is not advisable to suspend the lid from a weak superior rectus.

The three general types of ptosis operations are discussed and indications for each type are given. One operation in each category is described, including my method of levator resection done through the skin surface.

In general, a levator resection is indicated in a unilateral ptosis if there is any levator function whatsoever, or without levator function if there is strong binocular vision and fair or good function of the superior rectus. In bilateral ptosis, a levator resection is indicated if there is any levator function.

Suspension from the superior rectus (the Berke-Motais operation) is indicated in unilateral ptosis with complete paralysis of the levator, a fairly strong superior rectus, and absent or weak binocular vision. It is indicated in bilateral ptosis with complete levator paralysis and fair or good function of the superior recti.

Suspension from the frontalis is usually not the operation of choice in unilateral ptosis. It may be indicated in the occasional case with absent levator function and poor or absent function of the superior rectus, if the patient spontaneously uses his brow to elevate the lid. The chief indication for utilization of the frontalis is in bilateral ptosis with paralytic levators, especially if the superior recti are also weak.

The results in 162 ptosis operations are summarized, representative cases are photographically illustrated, and statistics are given in various categories of ptosis. The poorest results were obtained after levator resections in eyes with weak superior recti and the best after levator resections in eyes uncomplicated by other muscular anomalies. In the former group the percentage of good and excellent results was 56 percent, and in the latter group, it was 85 percent.

5 Bay State Road (15).

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EARLY MALIGNANT MELANOMA OF THE CHOROID*

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This study of early malignant melanoma of the choroid involving the posterior pole was undertaken in order to see whether information could be gleaned to facilitate an earlier clinical diagnosis. This material was limited to those cases with intact anterior segments which would have allowed unimpeded ophthalmoscopic examination. The clinical and pathologic files of the Episcopal Eye, Ear, and Throat Hospital have been utilized, together with the unsurpassed accumulation of malignant melanomas of the eye in the Registry of Ophthalmic Pathology at the Armed Forces Institute of Pathology.

GROUP I: FLAT MALIGNANT MELANOMAS OF THE CHOROID

This group is made up of the earliest lesions and thus presents the greatest clinical diagnostic difficulties. The accumulation of neoplastic cells has not yet produced obvious elevations of the lamina vitrea of the choroid.

The majority of the tumors in the 18 cases of this group appeared clinically as slight retinal elevations, usually gray in color, near the macula, and some were seen as slate-colored retinal detachments (five cases). The commonest symptom was blurring of vision. Diplopia was noted in one case, defective temporal vision in one, and faulty scleral transillumination was present in two cases. Symptoms had been noted for one month or less by four patients, for five weeks to 10 months by seven, for one to three years by four, and for five years or more by two. In one case, a large retinal detachment appeared four years after radical excision of the breast.

This group of early choroidal malignant melanomas was scrutinized with particular care to determine whether it would reveal any clues to aid in an earlier clinical diagnosis. Attention was finally focused upon two factors of definite interest.

Cystoid degeneration of the retina overlying the tumor has merited neither clinical nor pathologic discussion. Duke-Elder[1] mentioned it in passing, stating that it is found invariably in the retina overlying choroidal tumors. Although we cannot agree with this generalization, cystoid degeneration was the most striking pathologic change that we observed in these early choroidal tumors.

It was seen in 10 of the 18 cases, and in each of the 10 there were adhesions of varying degree between the tumor and the overlying retina (fig. 1). In the only other case with similar adhesions cystoid degeneration was absent, but extensive retinal gliosis was observed. In Case 1, the choriocapillaris and the overlying retina appeared normal.

Although the other seven cases did not show cystoid degeneration, in each of them serous exudation, varying in amount, separated the tumor from the retina.

Pigmentation is always commented upon by the clinician and has received a great deal of attention in pathologic discussion. It was found to be of no significance in these 18 cases. In none of them was there abnormal pigmentation within the retina. The retinal pigment epithelium showed various degrees of proliferation in half the cases, but this bore no relationship to tumor metabolism. The pigmentation of the tumor itself varied and yielded no clues for early diagnosis.

GROUP II: MALIGNANT MELANOMAS PRODUCING BULGE OF THE LAMINA VITREA

Forty-eight cases comprise this group in which the melanomas have increased in thickness and produced bulging of the lamina

* From the Department of Ophthalmic Pathology, Episcopal Eye, Ear, and Throat Hospital, and the Registry of Ophthalmic Pathology, The Armed Forces Institute of Pathology. Presented at the Wilmer Residents Association meeting, Baltimore, Maryland, April 27, 1953.

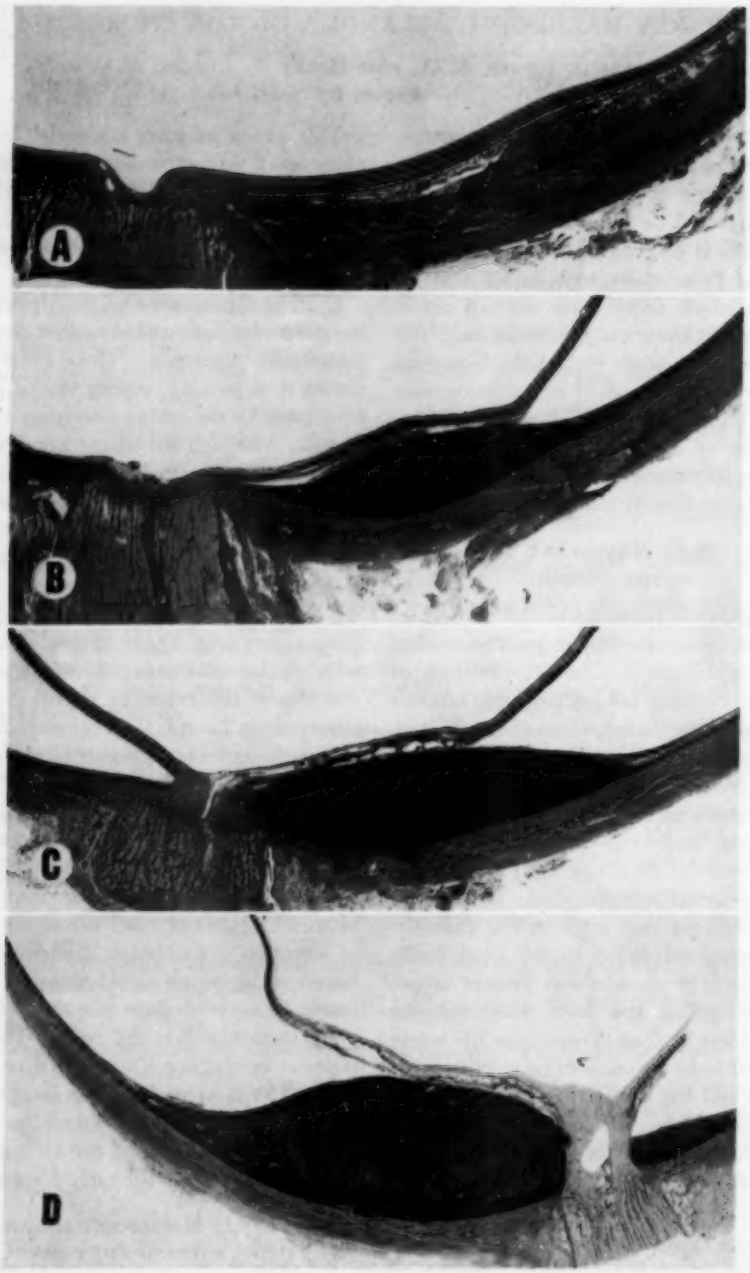

Fig. 1 (Rones and Linger). Flat malignant melanomas of the choroid. (A) In the earliest stage the tumor has produced slight elevation of the retina. (B) Serous exudation is seen between tumor and retina. (C and D) Cystoid degeneration is prominent in the retina which is attached to the tumor by adhesions. (AFIP Acc. 101543, ×13; AFIP Acc. 190255, ×13; AFIP Acc. 233002, ×13; AFIP Acc. 187798, ×8.)

vitrea but have not broken through this constraining barrier. The retinal detachment is of greater degree rather than of longer duration, as compared to Group I where some of the smaller melanomas were known to have been present for an even longer time.

The striking observation in this series is that subretinal serous exudation was absent in only two of these cases. In all the others there was fluid beneath the retina. The amount of exudate bore no relationship to the size of the tumor, for a number of the smaller tumors were accompanied by considerably more exudate than the larger ones.

Pigmentation was of greater interest in this series than in Group I. The pigment content of the tumor varied both in amount and distribution just as in the smaller tumors, but the pigment epithelium of the retina was more significantly altered with marked hyperplasia in 31 cases. In some the cells had scattered and formed clumps in the subretinal fluid, while in others the proliferated pigment cells had become confluent with those of the tumor itself. In only three cases was there migration of pigment into the retina.

Here also the relationship between cystoid degeneration of the retina and retino-neoplastic adhesions was striking. Twenty-two cases showed definite cystoid degenerative changes in the retina overlying the tumor (fig. 2), and in 20 of these there were well-marked adhesions between these structures. Conversely, in only four cases where adhesions were present was there no evidence of cystoid degeneration.

In these larger tumors, as might be expected, gliosis of the overlying retina was more frequent than in Group I, where it occurred only once. In Group II, it was found in 18 cases, in six of which it was associated with cystoid degenerative changes.

Group III: Malignant melanomas rupturing the lamina vitrea

In the 51 cases of this group the tumor has broken through the restraining barrier of the lamina vitrea. The dominant criterion in selecting these cases from several thousand advanced malignant melanomas of the choroid was that they still presented a diagnostic problem when observed ophthalmoscopically, and this implies an intact anterior segment with clear media. These 51 cases could be divided into three subgroups with respect to the destructive effect on the retina overlying the tumor.

In nine cases, the tumor had broken through the lamina vitrea and the overlying retina had undergone either gliosis or cystoid degeneration. Among these, two showed the early stages of invasion of the retina by the tumor and one, impingement of the tumor on the optic disc.

Subgroup 2 is made up of 26 cases in which the retina overlying the tumor was thinned and atrophic as a result of invasion (fig. 3-A) or pressure (fig. 3-B) or a combination of these factors. Serous exudation was always present, lifting adjacent areas of the retina away from the choroid. The atrophic retina can become so thinned that it is difficult to trace over the surface of the protruding tumor (fig. 4-A).

In the final subgroup of 16 cases, there was no retinal or fibrous covering of a portion of the tumor which protruded its naked aspect into the vitreous cavity where it could be observed with the ophthalmoscope (fig. 4-B).

Discussion of the material

Our purpose in studying this series of early tumors was to relate the size of the tumor, with due allowance for variations in speed of growth, to the various retinal phenomena which could be observed clinically by ophthalmoscopic examination. In the earliest tumors, the 18 cases of Group I, it was noted that in one case the choriocapillaris was still intact and that the overlying retina showed no morphologic alternation other than a slight elevation from its normal position. In a stage just beyond this, one of two events occurred—either the contour of the bulge remained in contact with the retina or

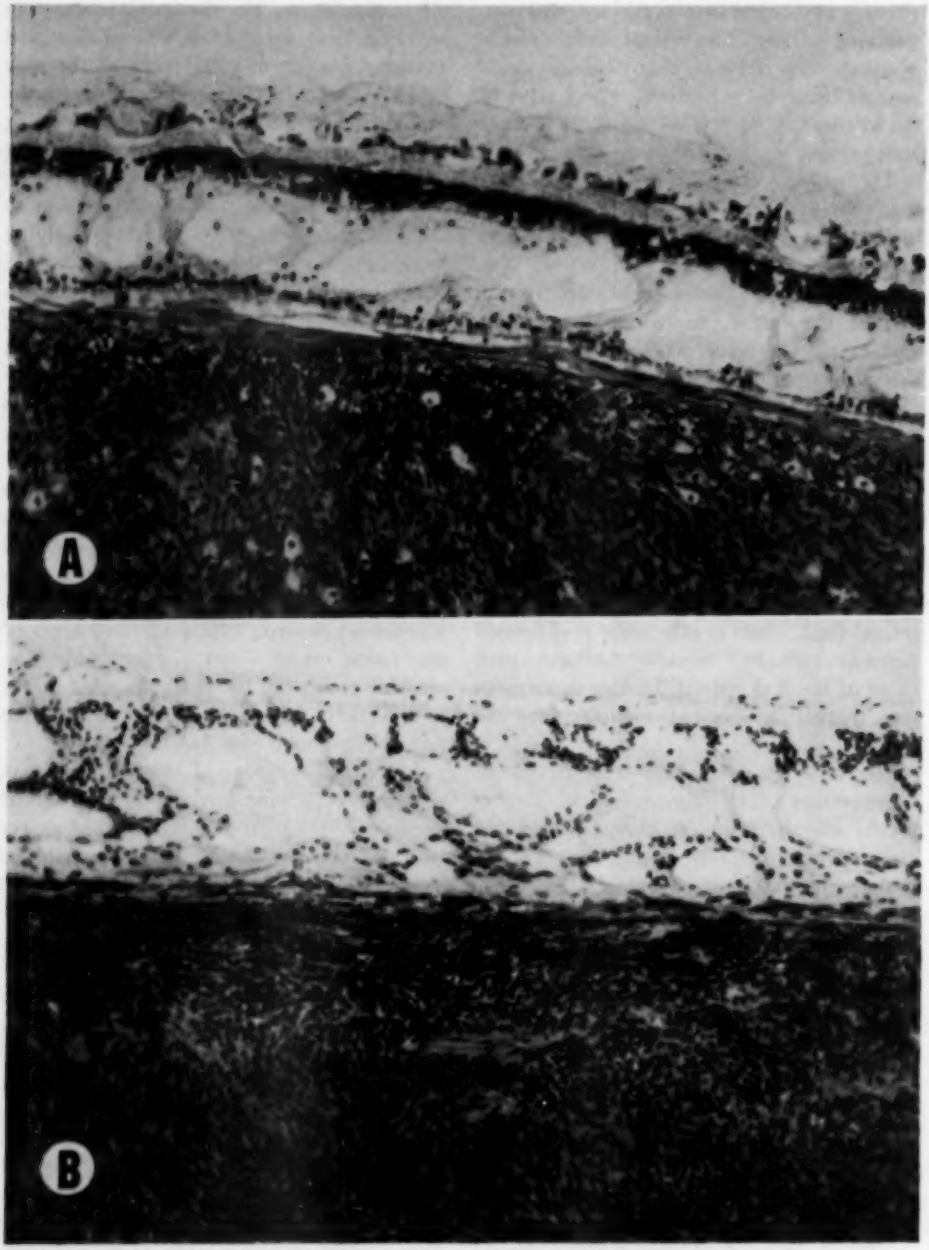

Fig. 2 (Rones and Linger). In further advanced malignant melanomas causing bulging of the lamina vitrea, cystoid degeneration is striking in the overlying retina. (AFIP Acc. 188419, ×115; AFIP Acc. 237133, ×115.)

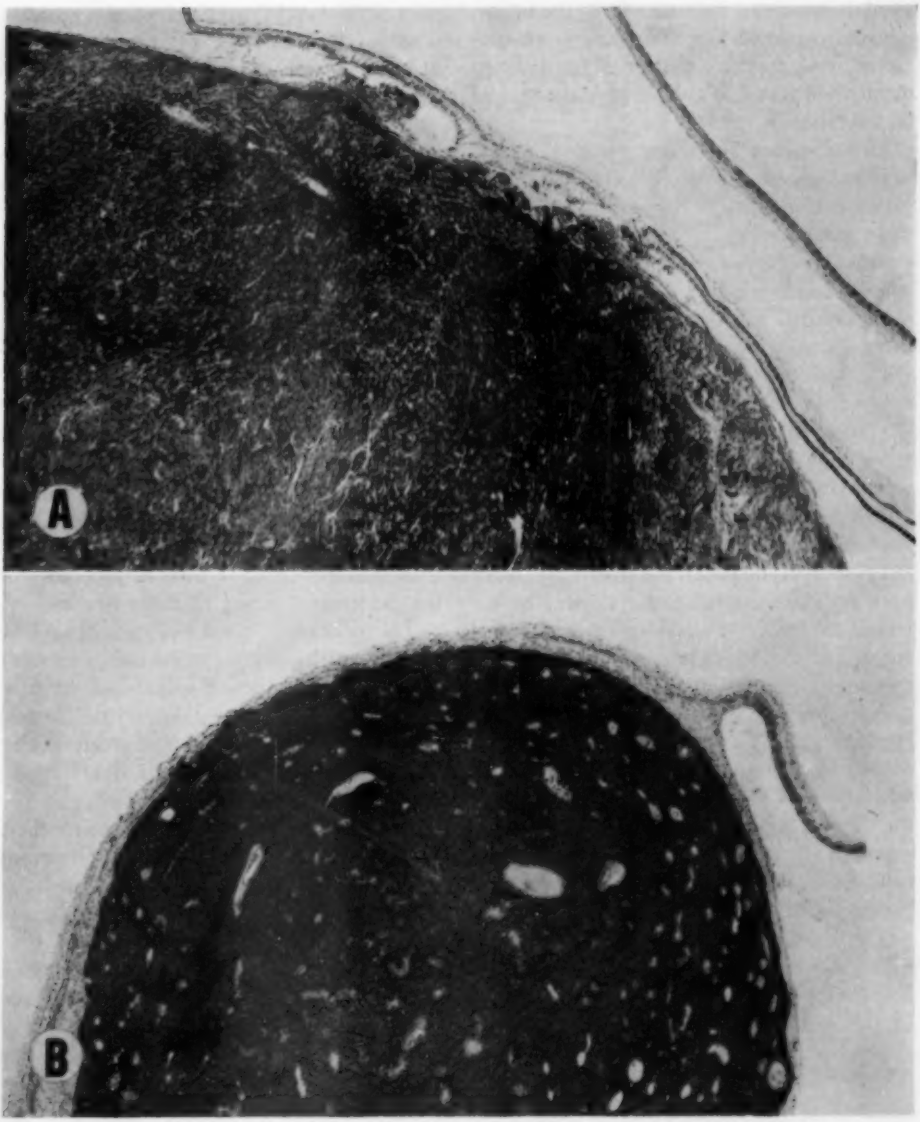

Fig. 3 (Rones and Linger). In malignant melanomas that have caused rupture of the lamina vitrea, the retina overlying the tumor is thin and atrophic from invasion (A) or pressure (B). (AFIP Acc. 176478, ×19; AFIP Acc. 206585, ×21.)

became separated from it by a layer of serous exudate. At points where the tumor and the retina remained in contact, extensive adhesions developed and the structure of the retina became altered.

Extensive cystoid degeneration was noted in 10 and retinal gliosis in one of the 11 cases with adhesions. However, where serous

exudate separated the retina from the tumor, apparently enough nutriment was supplied to the retina by this fluid to prevent the development of such visible morphologic changes of degeneration.

This does not imply that no exudate was present where adhesions were noted, but rather that in areas of direct contact between these tissues there was competition for the metabolites furnished by a limited blood supply. Beginning proliferation and necrosis of the retinal pigment epithelium were further evidence of this faulty vascularity.[2]

Pigment had not migrated into the retina either from the pigment epithelium or from the tumor itself.

In the second group of 48 cases, the melanomas were still confined to the choroid but were larger than those of Group I. There was considerable serous extravasation in all but two cases; however, the size of the tumor bore no relationship to the amount of the subretinal fluid, nor could any other observed factor be held accountable for the extent of this out-pouring of fluid.

Again the striking relationship between the contact of retina with tumor and the development of cystoid degeneration of the retina was noted. The proliferation and necrosis of the retinal pigment epithelium were more pronounced in this series, and the pigment cells were more widely dispersed throughout the subretinal serous exudate.

The character of the detachment of the overlying retina elicited our interest. No holes were visible either over the tumor or in the peripheral retina, where cystoid degeneration was noted with great frequency. The retinal contours varied depending upon the amount of underlying fluid and the presence of adhesions to the tumor.

The final group of 51 tumors that had ruptured the restraining barrier of the lamina vitrea and proliferated subretinally included nine with the mushroom type of growth, and in only two was there evidence of early invasion of the retina. In 26 cases the retina overlying the tumor was considerably thinned, either by pressure of the growing dome of the tumor or by digestion of the invading tumor cells. In the remaining 16 cases portions of the tumor were "naked" and the melanoma itself projected into the vitreous cavity. In all of these cases there were accompanying serous exudates beneath portions of the retina.

Clinical interpretation of the material

The clinician is interested in this pathologic material primarily as an aid in arriving at an earlier and more accurate diagnosis. The cases utilized in this study are of such a nature that little diagnostic information would be obtained from such accessory findings as elevated intraocular pressure, hemorrhagic or necrotic changes producing atypical inflammations, or atypical pigmentary degenerative changes in the eye. Studies with scleral transillumination offer little help because of the posterior position of these tumors.

The primary clinical evidence would be visual disturbances due to the involvement of the macula or adjacent areas, and the ophthalmoscopic interpretation of the retinal detachment. This material is of greatest value in the latter respect, as an adjunct to the direct ophthalmoscopic observation.

The problem is first to differentiate these early posterior neoplasms from spontaneous serous retinal detachments, and then, if possible, to separate them from nonmalignant lesions involving the same structures.

Serous detachment of the retina occurs peripherally in the great majority of cases, with a sudden onset and proportionate loss of vision. The retina is thrown into folds and the detachment has no sharply defined margins. A characteristic hole or "tear" in the retina is practically always noted on careful search. On the other hand, the retina over these early malignant melanomas of the choroid is smoothly drawn and does not show the tendency to wrinkle; furthermore, a hole in the retina is seldom seen.

In our cases considerable serous exudate frequently accompanied even small tumors,

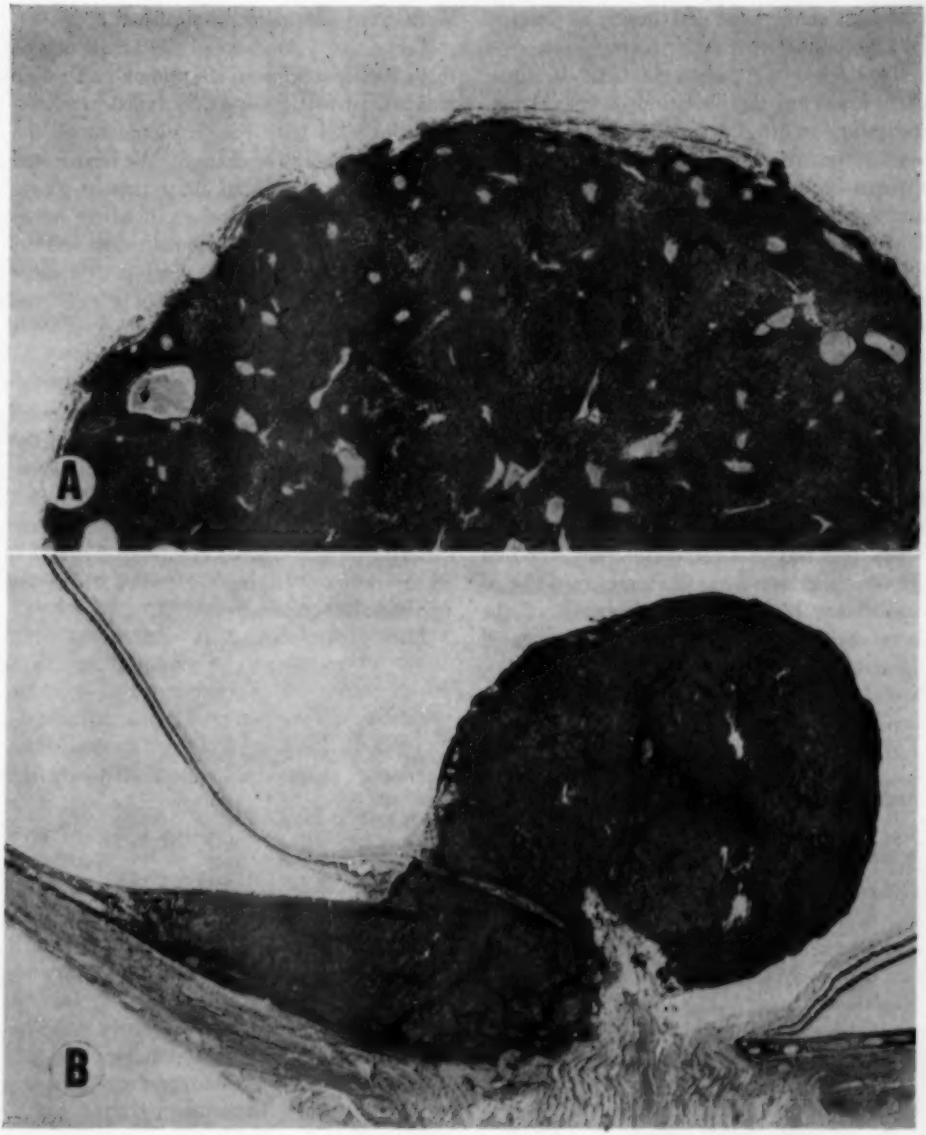

Fig. 4 (Rones and Linger). Malignant melanomas that protrude into the vitreous cavity are covered either by retina so thin that it can hardly be traced (A) or by none at all (B). (AFIP Acc. 170025; AFIP Acc. 189127, ×12.

and it is a well-known clinical fact that this subretinal fluid can shift its position, usually to the lower periphery, and in this way mask the true nature of the primary lesion. Such peripheral retinal detachments were described pathologically by Samuels[8] in 10 of 19 cases of melanoma studied, and they were frequently seen in our series. However, we

could not confirm the detachment of the pars ciliaris retinae reported by Klien.[4]

There was no pathologic basis in these early cases for the variation of retinal pigmentation so often described. The hyperplasia and necrosis of the retinal pigment epithelium occurs in both types of detachment and should produce the same effect. The solidity of the tumor, the extent of its pigmentation, and the albuminous content of its associated exudate probably have some influence on the character of the reflected light of the ophthalmoscope, and thus somewhat alter the appearance.

In Group III, where the naked tumor projected into the vitreous cavity, the irregularity of the surface and the presence of hemorrhages or new blood vessels should be of diagnostic significance. However, this study suggests that the cystoid degeneration of the retina overlying the tumor could be of importance in the clinical diagnosis of the very early choroidal melanomas if it could be detected ophthalmoscopically.

Terry and Johns[5] noted the frequency of the adhesions between the tumor and retina and it is surprising that they failed to associate adhesions with the development of the cystoid degenerative changes. We have noted cystoid degeneration of the retina in a case of hemangioma of the choroid where adhesions were present between the retina and the tumor, but we did not observe it in a large number of metastatic lesions of the choroid that we examined.

Summary

This is a study of 117 cases of malignant melanoma of the choroid which involved the posterior segment of the globe only. This selection was made on the basis of unimpeded ophthalmoscopic observation. The significant observation was that of cystoid degeneration in the retina overlying the tumor where the two were adherent.

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Ophthalmic Miniature

If paralysis of the orbicularis for the necessary length of time is ever accomplished, it will have to be by means of a drug which acts on the motor nerve endings when injected into the muscle. There is one drug which will do this—curare—but I have never dared to experiment with such a lethal weapon."

Lieut. Col. Henry Smith,
The Treatment of Cataract, Calcutta, Butterworth, 1928, page 92.

THE HISTOCHEMISTRY OF THE BASEMENT MEMBRANE OF THE CORNEA*

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INTRODUCTION

Since the Hotchkiss-McManus periodic-acid fuchsin stain was introduced to ophthalmology, Teng and Katzin[1] have observed that the corneal epithelium has a basement membrane, which can be distinguished from Bowman's membrane through the use of this stain. It is a continuous membrane which extends beyond the limbus and into the bulbar conjunctiva. It stains a striking violet color and has been demonstrated in a number of animals, including the steer. In the steer it is "fairly uniform, easy to see, and comparable to human eyes."[1] In our experiments we used beef corneas, and whenever possible confirmed our results with human corneas.

The significance of the basement membrane is stressed because of the work of Herrmann and Hickman[2] on the adhesive forces operating between Bowman's membrane and the corneal epithelium; the work of Cogan and Kinsey[3-6] relative to the permeability and hydration characteristics of the cornea and its limiting membranes; and the work of Gersh and Catchpole[7] on the depolymerization of the basement membrane of small blood vessels during rapid tumor growth.

Since there are no available methods for isolation of the basement membrane to make a direct chemical analysis, we conducted an investigation into the histochemical character of the basement membrane in order that a better understanding of the physiologic and pathologic character of the basement membrane might be obtained.

In this paper, our methods include only those histochemical reactions directly observable under the microscope.

EXPERIMENTAL

Beef eyes were preserved in a cooled thermos container and were used three to four hours after the animals had been slaughtered. Both human and beef eyes were used in all experiments except in the part dealing with enzyme reactions. In that part only beef eyes were employed.

LIPIDS

We started our experimental work with a search for fatty substances within the basement membrane. Because of the similarities in the physical properties of lipids as a group, sharp distinction between subgroups is extremely difficult in microscopic histochemistry. However, methods for unsaturated fatty acids are relatively specific for phospholipids and cholesterol esters because of the marked unsaturation of these compounds.[8]

In order to determine whether the basement membrane would stain with fat stains, the technique for Sudan IV and osmic acid described by Mallory[9] was used on frozen sections, on corneas fixed in formalin, and also on corneas fixed by Baker's method.[10] His method renders phospholipids insoluble by immersing small strips of tissue fixed in a solution of one-percent calcium chloride in formalin. In addition, Flemming's solution[9] was used as a fixative for sections later embedded in celloidin. Flemming's solution consists of osmic acid, chromic acid, and acetic acid.

Histochemical reactions for the identification of free aldehydes and ketones were used in both fresh frozen sections and celloidin embedded sections. This was done by sus-

* From the Eye-Bank Laboratory, aided by Grant B-153(C2) of the National Institutes of Health, Department of Health, Education, and Welfare, and the Dazian Foundation.

pending the tissue sections in Schiff's reagent for as long as 30 minutes. Schiff's reagent is made up of basic fuchsin, sodium sulfite, and hydrochloric acid. It forms highly colored addition complexes with aldehydes and ketones.[11]

Cholesterol and its esters can be identified by Schultz's method,[8, 12] which was applied to the basement membrane of the cornea. This test can be considered specific for all practical purposes.[8] In addition, phosphatides were sought by Baker's[10] modification of the Lorrain Smith method.[13] It is specific for lecithin, cephalin, and sphingomyelin and has been confirmed by Cain.[14]

Tests were applied for the identification of another type of phospholipid, namely, the lipid aldehyde. For this the Oster modification of the Feulgen reaction[15] and the plasmal reaction after Hayes were applied to fresh frozen sections (fig. 1). These reactions involve the action of mercuric chloride upon the acetal phosphatides, releasing higher fatty aldehydes.[11] The Feulgen reaction identifies specifically acetal phosphatides which are also called plasmalogens. Plasmalogens are glycero-phosphorylcolamine in a cyclic acetal linkage with one molecule of fatty aldehyde. There are two types possible,[16] depending upon whether the acetals are combined with the α carbons of glycerol or with the α' carbons.

The results of the above test are shown in Table 1.

As can be seen from Table 1, the basement membrane of the cornea stains with Sudan IV, osmic acid, and Feulgen reagent. From the fact that it does not stain with other lipid reagents, we can conclude that it does not contain cholesterol and its esters, the phospholipids, cephalin, lecithin, and sphingomyelin, or compounds containing free aldehydes and ketones in concentrations sufficient to give these reactions. Feulgen's plasmal reaction was positive according to Oster's modification and Hayes methods of identification.

For lipid extraction, whole corneas were suspended in 50 cc. of various lipid solvents as indicated in Table 2. They were suspended for four hours at room temperature. The controls were suspended in distilled water. The corneas were then rinsed in distilled water and placed in Bouin's solution. Routine paraffin embedding and staining techniques were employed with periodic-acid fuchsin stain. When butyl and amyl alcohols were used as lipid solvents in addition to the extraction and staining method outlined above, the Altmann Gersh freezing drying technique, formalin, and Bouin's solution were used as alternative fixing agents. This was done to make a comparative study of various fixative methods. Results are shown in Table 2.

The effect of lipid solvents on the basement membrane shows that n-propyl and isopropyl alcohols are the only ones tried that have any effect on the staining reaction. These alcohols do not cause a separation of the epithelium from the stroma. It is not concluded that these alcohols dissolve out the entire basement membrane but they seem to have interfered with the staining reaction.

TABLE 1

BASEMENT MEMBRANE STAINS FOR LIPIDS

Stain	Substance Stained	Reaction
Sudan IV	Tryglycerides, fatty acids, phospho-lipids	+
Osmic acid	Unsaturated fatty acids	+
Feulgen reaction	Plasmalogen	+
Schultz cholesterol test	Cholesterol and its esters	−
Dichromate method (Lorrain Smith's method)	Lecithin, cephalin, sphingomyelin	−
Schiff's reagent	Free aldehydes	−

TABLE 2

EFFECT OF SOLVENTS

At Room Temperature (24°C.) Fat Solvents—Absolute Chemically Pure	Stain with Periodic-Acid Fuchsin for Presence of Basement Membrane
1. Methyl alcohol	+
2. Methyl alcohol at 62°C.	+
3. Ethyl alcohol (96%)	+
4. Ethyl alcohol at 62°C. (96%)	+
5. N-Propyl alcohol	0
6. Iso-Propyl alcohol	0
7. N-Butyl alcohol	+*
	0
8. Tertiary butyl alcohol	+*
	0
9. N-amyl alcohol	+
10. Iso amyl alcohol	+
11. Acetone	+
12. Chloroform	+
13. Ether	+
14. Petroleum ether	+
15. Pyridine	+
16. Benzine	+

* Basement membrane stains intermittently.

Normal butyl and tertiary butyl alcohols do interfere irregularly with the staining of the basement membrane.

GLYCOGEN

In the attempt to determine whether the basement membrane contained glycogen, Best's carmine stain[9] was employed. In this method absolute alcohol is used to prevent the possible loss of glycogen by solution. Langhan's iodine stain was also used on other sections. The latter lacks specificity, however, and also stains amyloid and some protein substances.[8, 11] For polysaccharide sulfate compounds the Lison method[12] was employed. This involves the staining with Toluidin blue of mucoid compounds composed of polysaccharide esters of sulfuric acid.

OTHER CARBOHYDRATES

A test was made on the membrane for the presence of substances containing adjacent OH groups such as exists in many carbohydrates. This test consists of the addition of acetic anhydride with pyridine, and it involves a chemical reaction in which there is acetylation of adjacent OH groups which prevents the oxidation of these groups by

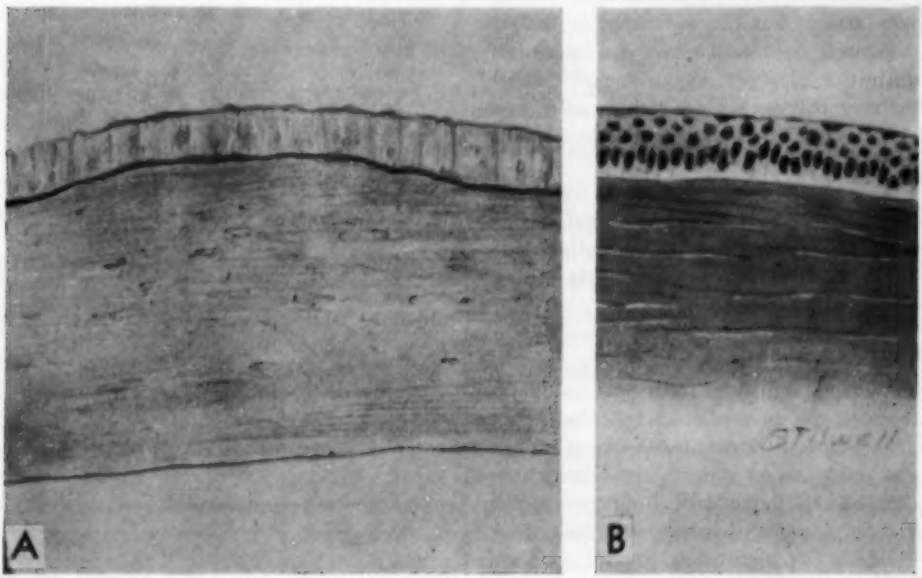

Fig. 1 (LaTessa, Teng, and Katzin). (A) Hayes' stain for plasmalogen in the basement membrane of rabbit cornea. (B) Normal rabbit cornea. (Hematoxylin-eosin.)

TABLE 3

STAINS FOR POLYSACCHARIDES

Stain	Substance Stained	Reaction
1. Periodic acid (Schiff's)	Polysaccharides and polysaccharidelike compounds	+
2. Best's carmine	Glycogen	−
3. Langhan's iodine	Glycogen, amyloid, lecithin, some protein substances	+
4. Lison's toluidin blue (Metachromasia)	Sulfated or other high molecular acidic polysaccharides	−

periodic-acid to an aldehyde linkage. Corneal sections were placed in acetic anhydride containing pyridine for 45 minutes and then the periodic acid fuchsin was applied. As a control, other sections treated with acetic anhydride were subsequently hydrolized with 1 N potassium hydroxide for 45 minutes. (Hydrolysis reverses acetylation and restores the adjacent hydroxyl groups.)

This method was suggested by McManus and Cason.[11] Although it is an accepted method for this analysis, the chemical basis of the reaction has not been demonstrated to the satisfaction of all observers. Results are shown in Table 3.

The stains for polysaccharides are less satisfactory, but the indication is that the membrane does not contain in demonstrable quantities, glycogen, hyaluronic acid, and/or the mucopolysaccharides giving rise to metachromasia. Langhan's iodine stain was positive but this test is not specific.

As a result of the acetylation test of McManus and Cason, we conclude that the basement membrane must contain adjacent hydroxyl groups. This is most likely a carbohydrate.

Reticulum

Wilder's reticulum stain* was used to determine the presence of reticulum fibers. This stain differentiates between collagen and reticulum by staining the former a rose color and the latter black. Both cross sections and flat sections of cornea were stained with Wilder's reticulum stain. Results are shown in Table 4.

The results obtained with Wilder's reticulum stain were quite interesting. By Wilder's reticulum stain we found an argyrophilic network in the basement membrane. It exists in a spongy form, and is composed of two to three layers of vacuoles. In the superficial layer the vacuoles are slightly larger than in the posterior layers and stain more deeply. The network of the vacuoles is made of more or less continuous fibers in an irregular pattern (figs. 2 and 3). The basement membrane loses its spongy appearance at the limbus and forms an irregular wavy fibrous structure with very little network. The argyrophilic network of fibers in the basement membrane is, we believe, reticular fibers.

Enzyme reactions

To observe the effects of enzymes upon the basement membrane, solutions of various enzymes in 0.9-percent sodium chloride were injected into beef corneas through the limbus. Then the corneas were incubated at 32°C. for four hours. Our purified enzyme preparation of trypsin* contained approximately 50 percent of $MgSO_4$. Salt-free chymotrypsin* and ribonuclease* were also used. Hyaluronidase† with an activity of 100 TRU/mg. was injected in the same way. Saliva was used as the source of ptyalin.

TABLE 4

STAIN FOR RETICULUM

Stain	Substance Stained	Reaction
Wilder's reticulum stain	Reticulum fibers	+

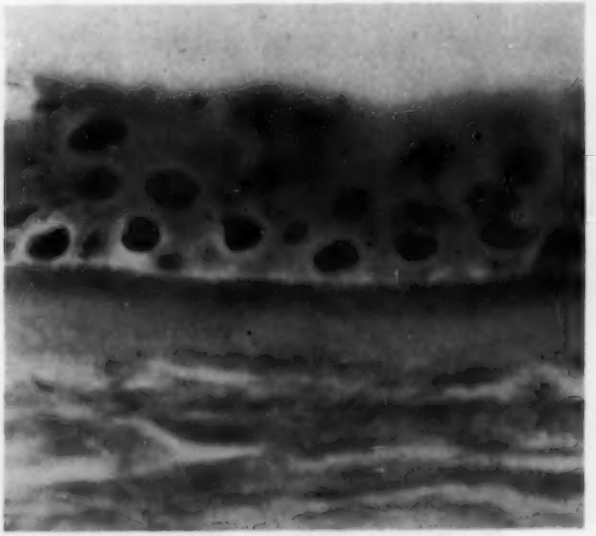

Fig. 2 (LaTessa, Teng, and Katzin). Reticular fiber network in the basement membrane of a human cornea. (Wilder's stain. Cross section.)

Sections were stained with periodic acid fuchsin.

Results are shown in Table 5.

The proteolytic enzymes, trypsin and chymotrypsin, abolish the staining reaction of the basement membrane. They also cause separation of the epithelium. Chymotrypsin was regularly effective on the basement membrane while the effect of trypsin was inconstant. Study of the sections showed that the basement membrane was actually destroyed by these enzymes. Ribonuclease, hyaluronidase, and saliva had no effect.

TABLE 5

EFFECT OF ENZYMES

Enzyme	Effect on Basement Membrane	Effect on Adhesion
*Trypsin (containing approx. 50% $MgSO_4$)	+	+
	0	
*Chymotrypsin	+	+
*Ribonuclease	0	0
*Hyaluronidase	0	0
Saliva (ptyalin 0.75 cc.)	0	0

* 10 mg. dissolved in 0.75 cc. injected into each beef cornea.

DISCUSSION

The Hotchkiss-McManus periodic acid fuchsin stain has been shown to be fairly specific for polysaccharides or polysaccharidelike compounds. The reaction involves the oxidation of adjacent hydroxyl groups or a hydroxyl group vicinal to an amino group, to an aldehyde. The aldehyde then stains violet with Schiff's reagent. Periodic-acid fuchsin may also stain similar chemical groups that are not polysaccharides, such as phospholipids. From the solubility and staining reactions it appears most likely that the basement membrane of the cornea contains both a polysaccharide complex and a phospholipid.

The fact that the basement membrane takes a positive reaction with the Feulgen reagent is considered fairly conclusive evidence that the basement membrane contains plasmalogen. Plasmalogen is a phospholipid. Plasmalogens are generally soluble in most lipid solvents particularly in chloroform and cold ethanol.[18] After the corneas were ex-

* Obtained from Worthington Biochemical Laboratories, Freehold, New Jersey.

† Obtained from Treemond Company, New York, New York.

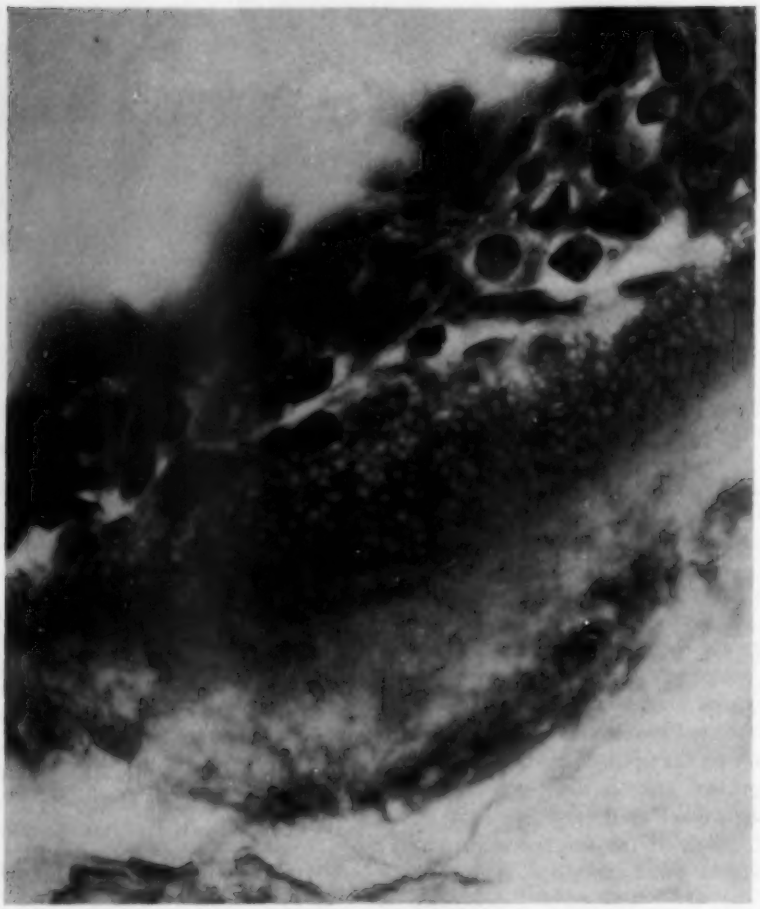

Fig. 3 (LaTessa, Teng, and Katzin). Reticular fiber network in the basement membrane of a human cornea. (Wilder's stain. Flat section.)

tracted with these lipid solvents, a positive stain with periodic-acid fuchsin could still be obtained. Hence, it is believed that the residual stain was due to a polysaccharide complex.

As confirmation of the fact that plasmalogen found in the basement membrane was removed by certain lipid solvents, the Feulgen reaction applied after extraction of the cornea with these lipid solvents was found to be negative. Furthermore, the separation between the epithelium and stroma occurred between the basement membrane and Bowman's membrane. This suggests that the plasmalogen may have some adhesive property, cementing the cells to their basement membrane.

Insofar as polysaccharides are concerned, acetic anhydride is thought to prevent oxidation of adjacent hydroxyl groups by acetylation, but hydrolysis with potassium hydroxide restores the hydroxyl group and with it the characteristic ability to stain with periodic-acid fuchsin. The significance of this is that there must be somewhere in the chemical structure of the basement mem-

brane, adjacent hydroxyl groups. Adjacent hydroxyl groups are most commonly found in polysaccharides and other carbohydrates.

The effect of proteolytic enzymes on the basement membrane is very interesting. It is possible that these enzymes have a lytic effect on the reticular structure that has been demonstrated by Wilder's stain. Our interpretation would be that after the enzyme activity has affected the reticular network the phospholipid and polysaccharide components are lost by solution during the subsequent tissue staining processes.

The natural conclusion would be that the protein and polysaccharide components of the basement membrane exist as muco- or glycoproteins, which are complexes of acetyl glucosamine with protein. The protein component of the basement membrane appears to be reticular in nature.

Friedenwald, Buschke, and Crowell,[16] in their experimental study upon injury to the corneal epithelium, found plasmal present in the basal layer. After injury to the epithelial cells they found a substance exuded into the wound which was lipoid soluble, binds silver, and was identified at least in part as plasmal.

Our experimental findings on the basement membrane of the cornea differ from the earlier findings of Gersh and Catchpole,[7] who worked with other organs, in two respects: namely, the existence of a spongy pattern of reticular fibers in the basement membrane of the cornea, and the demonstration of plasmalogen.

Conclusions

1. The basement membrane contains plasmalogen.

2. Separation or loss of cohesion between the epithelium and the basement membrane is effected by various lipid solvents indicating a lipoid layer which helps maintains adhesion. (Plasmalogen may contribute to the adhesive properties.)

3. The basement membrane contains reticular fibers.

4. The basement membrane is destroyed by the proteolytic enzymes, trypsin and chymotrypsin.

5. The nonlipid portion of the basement membrane probably contains a muco- or glycoprotein.

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EXOTROPIA WITH BILATERAL ELEVATION IN ADDUCTION*

Part II. Surgery

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It is the purpose of this paper to discuss the results of surgical intervention in my series of cases of exotropia with bilateral elevation in adduction. The results in 54 cases will be presented in tabular form. Cases will be presented to demonstrate corrections obtained with various types of surgery on the horizontal muscles alone and in combination with recession of the inferior oblique muscles. The effect of the various types of surgery on the position of the eyes in the straight-up and straight-down directions of gaze will be stressed.

Case reports

In the following two cases a bilateral recession of the lateral rectus muscles was done.

Case 1

J. W., aged 10 years, came to the Infirmary in January, 1951, with a history of turning out of both eyes as long as he could remember. Refraction with atropine cycloplegia was: O.D., −1.5D. sph. ⌒ +1.5D. cyl. ax. 115° = 20/20; O.S., −0.25D. sph. ⌒ +1.25D. cyl. ax. 75° = 20/20.

Preoperative examination (fig. 1A) revealed 30 to 40 degrees of exotropia for distance and upward gaze and straight eyes for near and downward gaze with stereopsis. The near-point of convergence was 50 mm. There was bilateral elevation in adduction with diagnostic vertical measurements. The finding of an exotropia for distance and upward gaze and straight eyes for near and down indicated that there was an apparent overaction of the lateral recti (divergence excess). Note that the exophoria measurements for near were greater than the exotropia measurements for distance (fig. 1A).

At surgery on May 20, 1951, a bilateral seven-mm. recession of the lateral rectus muscles was done. Postoperative examination on August 22, 1951, three months after surgery (fig. 1B) revealed straight eyes for distance and near with and without glasses. Ten degrees of exotropia remained looking up, although the eyes could be straight a good deal of the time in this position. The convergence near-point was 45 mm. and stereopsis was present. The bilateral elevation in adduction was much improved although the right hypertropia measurements had increased slightly.

Re-examination on May 23, 1952, one year after surgery, revealed that the eyes were now straight in all positions with much

Fig. 1A (Urist). Case 1, preoperative examination.

a. Straight for near without glasses (stereopsis present for near).

b. Forty degrees right exotropia for distance. (Note cover measurements only 45 prism diopters; patient is looking over the camera.)

c. Near-point of convergence is 50 mm.

d. Slight elevation of the left eye in adduction.

e. Eyes level on gaze to the left (elevation in adduction seen clinically).

f. Thirty-five degrees of left exotropia looking up.

g. Eyes straight looking down.

* From the Department of Ophthalmology of the Illinois Eye and Ear Infirmary, University of Illinois College of Medicine, Chicago. Part I of this paper appears in The Journal, **38**:58 (July) 1954.

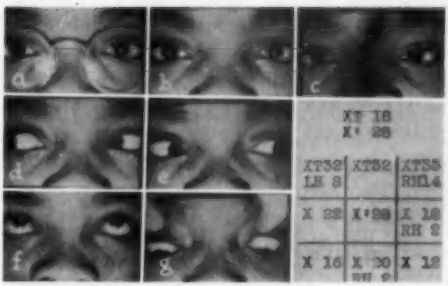

Fig. 1B (Urist). Case 1, postoperative examination, three months after surgery consisting of a bilateral seven-mm. recession of the lateral rectus muscles.

a. Straight for distance (and near) with glasses (stereopsis present).

b. Straight for distance (and near) without glasses (stereopsis present).

c. Near-point of convergence is 45 mm.

d. and e. Eyes level on lateral gaze; no limitation in abduction.

f. Straight looking up.

g. Straight looking down.

improvement in the vertical cover measurements.

Prism cover measurements were: X 12; X' 22 prism diopters.

	X 24, LH 4	X 23, RH 1	X 16, RH 6	
Right	X 10	X 22	X 8	Left
	X 8	X 12	X 8	

The bilateral seven-mm. recession of the lateral rectus muscles corrected the exotropia for distance and upward gaze without overcorrecting the previously straight eyes for near and looking down. No limitation in abduction was present following this amount of recession. With the correction of the exotropia in all positions the bilateral elevation in adduction disappeared and the vertical cover measurements became much improved.

Case 2

E. J., aged nine years, came to the Infirmary on September 9, 1948, with a history of his eyes turning out since birth. Refraction with atropine cycloplegia was: O.D., −6.5D. sph. ⌒ +2.75D. cyl. ax. 105° = 20/50; O.S., −6.0D. sph. ⌒ +2.75D. cyl. ax. 85° = 20/40.

Examination on December 22, 1949 (fig. 2A), revealed about 25 degrees of exotropia for distance and upward gaze. The eyes could be straight for near, although an exotropia was present a good deal of the time for near. There was 25 degrees of exotropia looking down and a near-point of convergence of 100 mm. Bilateral elevation in adduction was present with diagnostic vertical measurements. Alternating sursumduction was present with the delayed cover test.

At surgery on January 5, 1950, a bilateral seven-mm. recession of the lateral rectus muscles was done. Postoperative examination on July 30, 1950, six months after surgery (fig. 2B) revealed straight eyes for distance and upward gaze while the eyes remained straight for near. The near exophoria measurements were reduced from 50 prism diopters to 20 prism diopters. The near-point of convergence and the exotropia looking down were not improved. Stereopsis was present for distance and near. There was no limitation in abduction.

This case demonstrated beautifully that the lateral rectus muscles, when operated on, have their greatest effect on the distance and

Fig. 2A (Urist). Case 2, preoperative examination.

a. Eyes straight for near without glasses.

b. Twenty-five degrees of right exotropia for distance.

c. Eyes straight for near with glasses.

d. Elevation of the left eye in adduction.

e. Slight elevation of the right eye in adduction (seen well clinically).

f. Near-point of convergence 100 mm. with glasses.

g. Thirty degrees of right exotropia looking up.

h. Twenty-five degrees of right exotropia looking down.

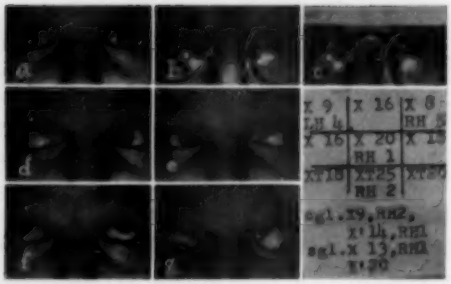

Fig. 2B (Urist). Case 2, postoperative, six months after a bilateral seven-mm. recession of the lateral rectus muscles.

a. Straight for distance without glasses.

b. Near-point of convergence 100 mm. with glasses.

c. Straight for near with glasses; has stereopsis.

d. and e. Eyes level on lateral gaze.

f. Eyes straight on upward gaze.

g. Remaining 25 degrees of left exotropia looking down.

straight-up positions of gaze. The bilateral seven-mm. recession of the lateral rectus muscles was sufficient to correct the greater exotropia for distance and upward gaze, but insufficient to correct the lesser exotropia looking down and the remote near-point of convergence. A six-mm. resection of one medial rectus may have corrected the exotropia looking down and improved the near-point of convergence as in Case 3.

In the following case a bilateral recession of the lateral rectus muscles and a unilateral resection of a medial rectus muscle was done.

Case 3

C. J., aged nine years, came to the Infirmary in 1949 with a history of the eyes turning out since birth. Refraction with atropine cycloplegia was: O.D., −3.75D. sph. ⌒ +0.75D. cyl. ax. 85° = 20/30; O.S., −0.25D. sph. ⌒ +0.25D. cyl. ax. 145° = 20/30.

Examination on September 26, 1949 (fig. 3A), revealed about 45 degrees of exotropia for distance and upward gaze and about 20 degrees to 25 degrees of exotropia for near and downward gaze. The eyes could be straight for near with a remote near-point of convergence when stimulated by the taking of photographs. Otherwise, when examined in the clinic, the eyes were always divergent with no near-point of convergence. Bilateral elevation in adduction was present, more on the right, with diagnostic vertical cover measurements. The delayed cover test showed alternating sursumduction with bilateral wheel rotation in. No fusion could be found.

At surgery, on October 27, 1949, a bilateral seven-mm. recession of the lateral rectus muscles and a six-mm. resection of the right medial rectus muscle was done. Postoperative examination on June 16, 1950, eight months after surgery (fig. 3B) revealed straight eyes for distance and upward gaze and for near and downward gaze. The near-point of convergence was 60 mm. The bilateral elevation in adduction was much

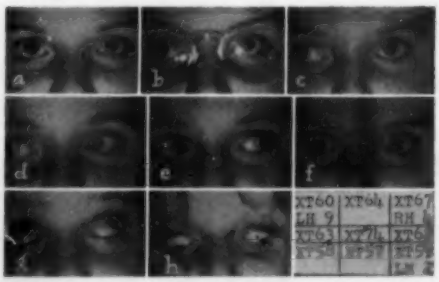

Fig. 3A (Urist). Case 3, preoperative examination.

a. Eyes straight for near (only found when being photographed, divergent at other times when examined in the clinic). Cover measurements are XT 74 prism diopters, right hyperphoria three prism diopters.

b. Twenty degrees of right exotropia for near with glasses.

c. Fifty degrees of right exotropia for distance without glasses. Cover measurements are XT 71 prism diopters, right hyperphoria four prism diopters.

d. Elevation of the left eye in adduction.

e. Slight elevation of the right eye in adduction (seen better clinically).

f. Near-point of convergence 120 mm. (found only when being photographed, otherwise no near-point could be elicited).

g. Forty-five degrees of left exotropia looking up.

h. Twenty-five degrees of left exotropia looking down.

improved. Only a trace of the alternating sursumduction and bilateral wheel rotation in was present.

The bilateral recession of the lateral rectus muscles was sufficient to correct the overaction of these muscles as manifested by the greater exotropia for distance and upward gaze. The six-mm. resection of the right medial rectus was sufficient to correct the underaction of the medial rectus as manifested by the smaller exotropia for near and downward gaze and the remote near-point of convergence.

With the correction of the exotropia the bilateral elevation in adduction, wheel rotation, and alternating sursumduction have all improved demonstrating that they most likely were secondary to the dissociation of the eyes by the exotropia.

In the following case the operation performed was a unilateral resection of a medial rectus and recession of a lateral rectus.

CASE 4

I. D., aged 15 years, came to the Infirmary on October 3, 1949, with a history of her eyes turning out since birth. She had double

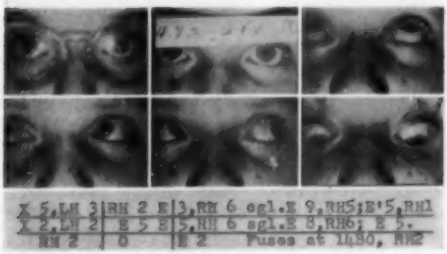

Fig. 3B (Urist). Case 3, postoperative examination, three months after a bilateral seven-mm. recession of the lateral rectus muscles and a six-mm. resection of the right medial rectus muscle.

a. Eyes straight for near with glasses (has second-grade fusion).

b. Eyes straight for distance without glasses.

c. Eyes straight looking up.

d. Slight elevation of the left eye in adduction; no limitation of the right eye in abduction.

e. Slight elevation of the right eye in adduction; no limitation of the left eye in abduction.

f. Eyes straight looking down (apparent left exotropia in photograph).

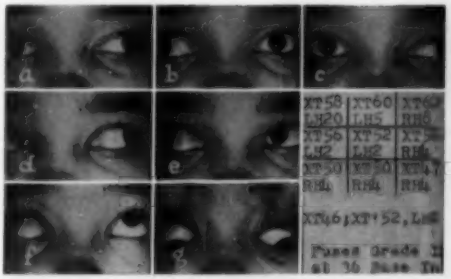

Fig. 4A (Urist). Case 4, Preoperative examination.

a. Forty-five degrees left exotropia for distance.

b. Forty-five degrees right exotropia for near.

c. No convergence near-point obtainable (a good near-point could be obtained with —3.0D. lenses).

d. Elevation and limitation of the right eye in adduction.

e. Elevation and limitation of the left eye in adduction.

f. Fifty degrees right exotropia looking up.

g. Twenty-five degrees left exotropia looking down.

vision at times. Refraction with atropine cycloplegia was: O.D., +0.5D. cyl. ax. 115° = 20/20; O.S., −1.25D. sph. ⊃ +0.5D. cyl. ax. 80° = 20/20.

Examination on May 15, 1950 (fig. 4A), revealed about 45 degrees of exotropia for distance and upward gaze although the cover measurements showed about half this amount, about 45 degrees of exotropia for near and 25 degrees looking down. No near-point of convergence could be obtained yet when minus 3.0D. lenses were placed before the eyes there was second-grade fusion at 36 diopters base-in on the major amblyoscope and a remote near-point of convergence could be obtained. Bilateral elevation and limitation in adduction, especially of the left eye, was present, with diagnostic vertical measurements.

At surgery on June 15, 1950, a five-mm. resection of the right medial rectus and an eight-mm. recession of the right lateral rectus were done. Postoperative examination on April 15, 1951, 10 months after surgery, revealed (fig. 4B) that for distance the eyes could be straight and also could have 20 degrees of exotropia a good deal of the time.

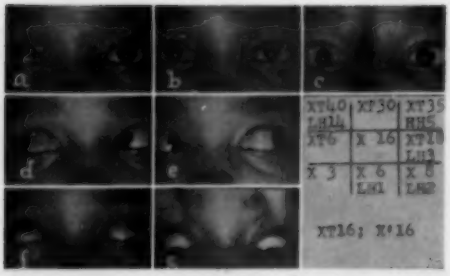

Fig. 4B (Urist). Case 4, postoperative examination, six months after a five-mm. resection of the right medial rectus and an eight-mm. recession of the right lateral rectus.

a. Twenty degrees of right exotropia for distance (can be straight for distance).

b. Eyes straight for near; has stereopsis at 0.

c. Near-point of convergence is 50 mm.

d. Slight elevation of the left eye in adduction with no limitation of the right eye in abduction.

e. Eyes level on gaze to the left; no limitation in adduction.

f. Twenty-five to 30 degrees of left exotropia looking up.

g. Eyes straight looking down.

There was 30 degrees of exotropia on upward gaze. The eyes were straight for near and looking down with stereopsis. The near-point of convergence was 50 mm.

The elevation and limitation of the right eye in adduction were corrected and there was improvement in the vertical measurements. Slight elevation and limitation of the left eye in adduction remained. No limitation in abduction of the right eye was seen following the eight-mm. recession of the lateral rectus muscle.

The five-mm. resection of the right medial rectus was sufficient to correct the underaction of the medial rectus muscles as manifested by the lesser exotropia for near and downward gaze. The unilateral eight-mm. recession of the right lateral rectus was insufficient to correct the greater exotropia for distance and upward gaze.

A bilateral recession of the lateral rectus muscles along with the resection of a medial rectus would have been the surgery to do as in Case 3. For reoperation, a seven to eight-mm. recession of the left lateral rectus muscle is indicated to correct the exotropia for distance and upward gaze.

In the following case a bilateral recession of the lateral rectus muscles and a bilateral resection of the medial rectus muscles was done in two operative procedures.

CASE 5

L. V., aged five years, came to the Infirmary on June 10, 1947, with a history of the eyes turning out since birth. Refraction with atropine cycloplegia was: O.D., −1.0D. sph. ⌒ +2.0D. cyl. ax. 90° = 20/70; O.S., +0.5D. sph. ⌒ +1.0D. cyl. ax. 90° = 20/30.

Preoperative examination (fig. 5A) revealed about 35 degrees of exotropia for distance and upward gaze, about 10 degrees for near, and about 20 degrees on downward gaze. The convergence near-point was

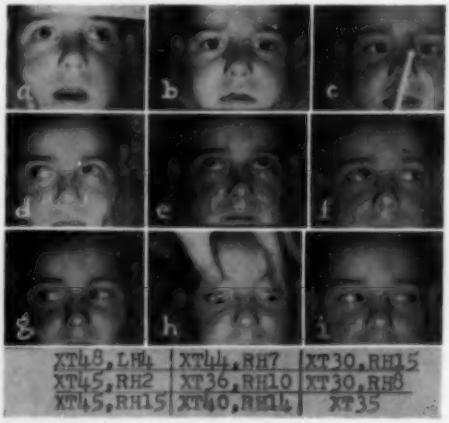

Fig. 5A (Urist). Case 5, preoperative examination.

a. Thirty-five degrees left exotropia for distance. (Note cover measurements are only XT 34 prism diopters, RH 15 prism diopters.)

b. Five to 10 degrees right exotropia for near.

c. Near-point of convergence is 60 mm.

d. Elevation of the left eye looking up and to the right.

e. Thirty-five degrees right exotropia looking up.

f. Elevation of the right eye on looking up and to the left.

g. Slight elevation of the left eye in adduction (seen well clinically).

h. Twenty degrees left exotropia looking down.

i. Slight elevation of the right eye in adduction (seen well clinically).

60 mm. There was elevation in adduction with diagnostic vertical measurements. Fusion could not be obtained.

Surgery on August 14, 1947, consisted of a 10-mm. recession of the right lateral rectus. Postoperative examination on October 16, 1947, two months after surgery (fig. 5B) revealed about 35 degrees of exotropia for distance and upward gaze, 25 degrees of exotropia for near and downward gaze, and a remote convergence near-point.

Bilateral elevation in adduction was present which was much greater for the left eye, yet, only a right hypertropia could be found with cover measurements (amblyopic eye hypertropia). Fusion was found on the major amblyoscope at 34 prism diopters base-in with a right hypertropia of 11 prism diopters.

The exotropia for near seemed to increase following the recession of the right lateral rectus (compare fig. 5A-b with fig. 5B-b). This demonstrated the marked effect of the innervational factors on the squint in these cases. (In this case the child feared more surgery.)

Note that following such a large recession there was no limitation of the right eye in abduction. The cover measurements had decreased for distance however. The near-point of convergence was remote at this time which showed the variability of this finding since it was 60 mm. before surgery.

It was felt that more surgery should be done both to the medial and lateral rectus muscles because of the large exotropia remaining in the straight-up and straight-down positions of gaze.

Reoperation on February 11, 1948, consisted of an eight-mm. resection of the left medial rectus and a five-mm. recession of the left lateral rectus along with a five-mm. resection of the right medial rectus. Postoperatively the eyes were straight for distance and for near. The mother was not pleased with the result because of the marked upshoot of the adducted eye when the child looked up at her.

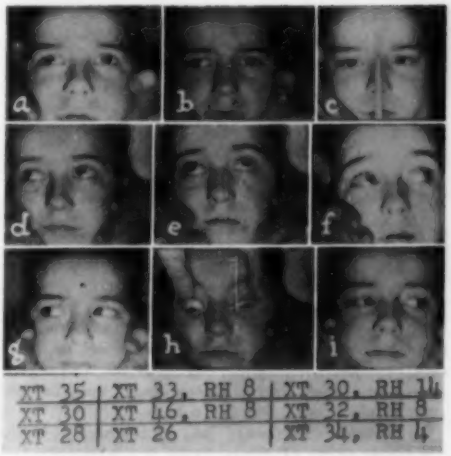

Fig. 5B (Urist). Case 5, postoperative examination, two months after a 10-mm. recession of the right lateral rectus muscle.

a. Thirty-five degrees of exotropia with 10 degrees of hypertropia of the right eye for distance. (Cover measurements only XT 25, RH 12 prism diopters.)

b. Twenty-five degrees right exotropia for near (Patient emotionally upset).

c. Near-point of convergence is remote.

d. Elevation of the left eye on gaze up and to the right.

e. Thirty-five degrees of exotropia looking up.

f. Elevation of the right eye on gaze up and to the left.

g. Slight elevation of the left eye in adduction (more obvious clinically); no limitation of the right eye in abduction.

h. Twenty-five degrees of left exotropia looking down.

i. Slight elevation of the right eye in adduction (more obvious clinically).

Examination on January 11, 1951, three years after surgery, revealed straight eyes for distance and upward gaze and for near and downward gaze with stereopsis (fig. 6). This was a functional cure of the exotropia. A trace of the bilateral elevation still remained but no vertical deviation was found with cover measurements.

This case demonstrated that the vertical deviations were secondary to the exotropia since lateral muscle surgery allowed good amplitude of fusion to develop with subsequent correction of the dissociated vertical deviations. The parents were pleased at this

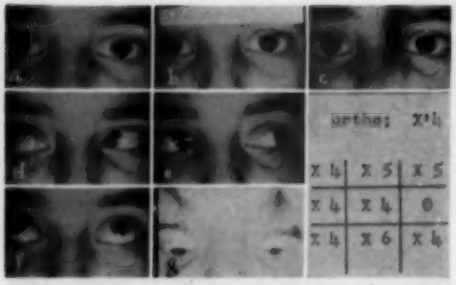

Fig. 6 (Urist). Case 5, postoperative examination, three years after reoperation consisting of an eight-mm. resection of the left medial rectus, five-mm. resection of the right medial rectus, and a five-mm. recession of the left lateral rectus muscles.

a. Straight for distance.
b. Straight for near; has stereopsis.
c. Near-point of convergence is 80 mm.
d. Slight elevation of the left eye in adduction.
e. Slight elevation of the right eye in adduction.
f. Straight looking up.
g. Straight looking down.

time because the upshoot in adduction had improved so much that it was not visible to them any more. Lateral muscle surgery corrected a right hypertropia of 15 prism diopters straight ahead and a right hypertropia of 15 prism diopters in levo-elevation.

In the following case a resection of a medial rectus muscle was done to correct a postoperative exotropia.

Case 6

A. B., aged five years, came to the Infirmary on November 16, 1945, with a history of the right eye turning in since 15 months of age. Refraction with atropine cycloplegia was: O.D., +1.25D. sph. = 20/20; O.S., +1.0D. cyl. ax. 80° = 20/20.

Preoperative examination on August 9, 1946, revealed 15 degrees of esotropia for near. The eyes could be straight at times. Cover measurements were: cgl., ET 30 prism diopters, ET' 30 prism diopters; sg. ET 37 prism diopters, ET' 40 prism diopters.

The near-point of convergence was 30 mm. Since the eyes were straight a good deal of the time, the attending surgeon felt that a small amount of surgery would be sufficient to help keep the eyes straight.

Surgery on September 9, 1946, consisted of a four-mm. recession of the right medial rectus and a four-resection of the right lateral rectus. Following this surgery, an exotropia developed.

Postoperative examination on June 4, 1947 (fig. 7A), nine months after surgery revealed about 25 degrees of exotropia for distance and upward gaze, 10 degrees of exotropia for near, and a near-point of convergence of 30 mm. The eyes could be straight for near and at such times there was second-grade fusion. The eyes were straight looking down. Marked bilateral elevation in adduction was present with small diagnostic vertical cover measurements. The attending surgeon felt that the medial rectus had slipped and should be reoperated.

On November 2, 1947, the right medial rectus muscle was exposed and found to be exactly where placed at the previous opera-

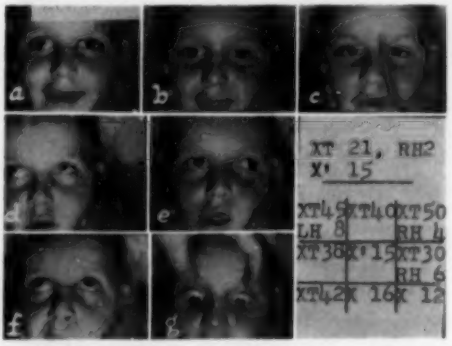

Fig. 7A (Urist). Case 6, postoperative examination, nine months after a four-mm. recession of the right medial rectus and a four-mm. resection of the right lateral rectus muscles.

a. Twenty-five degrees of exotropia with 10 degrees of hypertropia of the right eye for distance. (Cover measurements only XT 21 prism diopters.)
b. Ten degrees of right exotropia for near. (Can be straight for near and has second-grade fusion.)
c. Near-point of convergence is 30 mm.
d. Marked elevation of the left eye in adduction.
e. Marked elevation of the right eye in adduction.
f. Thirty-five degrees of right exotropia looking up.
g. Eyes straight looking down.

tion. Four mm. of the muscle were resected and it was advanced to the old insertion, five mm. from the limbus.

Postoperative examination on December 21, 1949, two years after reoperation (fig. 7B) revealed straight eyes for distance and near in the central field. The patient had stereopsis. An exotropia was present for distance at times. There was about 35 degrees of exotropia looking up and about 15 to 20 degrees of esotropia looking down. The near-point of convergence was 25 mm. Marked bilateral elevation in adduction was present with larger vertical measurements than before reoperation.

After only a small amount of surgery for an intermittent esotropia, a large overcorrection was produced which could be classified as an exotropia with bilateral elevation in adduction. This indicated that overaction of the lateral rectus muscles had developed manifested by the large exotropia present for distance and upward gaze. The good near-point of convergence and the straight eyes looking down indicated that the medial rectus had not slipped. This was confirmed at surgery.

After reoperation, the eyes became straight in the central field. This demonstrated that the overacting lateral rectus muscles (20 degrees of exotropia looking up) and the overacting medial rectus muscles (20 degrees of esotropia looking down) had reached an equilibrium in the central field so that the eyes could be straight for distance and near with stereopsis.

A bilateral recession of the lateral rectus muscles would probably have straightened the eyes in all positions without causing an esotropia looking down. The parents were not pleased with the result because of the large exotropia which was present for distance a good deal of the time and always when the child looked up at them.

In the following case surgery consisted of bilateral recession of the lateral rectus muscles along with bilateral recession of the inferior oblique muscles.

Fig. 7B (Urist). Case 6, postoperative examination, two years after reoperation consisting of a four-mm. resection and advancement of the right medial rectus muscle to five-mm. from the limbus.

a. Straight for distance; can be divergent for distance at times.

b. Straight for near; has third-grade fusion.

c. Near-point of convergence is 25 mm.

d. Marked elevation of the left eye in adduction.

e. Marked elevation of the right eye in adduction.

f. Thirty-five degrees of left exotropia looking up.

g. Fifteen to 20 degrees of right esotropia looking down.

Case 7

F. P., aged seven years, came to the Infirmary in July, 1944, with a history of the eyes turning out at times for distance. Refraction with atropine cycloplegia was: O.D., -2.25D. sph. $\bigcirc$ $+4.0$D. cyl. ax. 100° = 20/40; O.S., -2.25D. sph. $\bigcirc$ $+5.0$D. cyl. ax. 90° = 20/40.

Preoperative examination on August 10, 1949 (fig. 8A), revealed about 15 to 20 degrees of exotropia for distance, 25 to 35 degrees of exotropia looking up, straight eyes for near and looking down, and a convergence near-point of 50 mm. No fusion could be elicited. There was marked bilateral elevation in adduction with large diagnostic vertical measurements. The service diagnosed divergence excess and bilateral overaction of the inferior obliques due to bilateral paresis of the superior rectus muscles.

At surgery on September 10, 1949, a bilateral 8.5-mm. recession of the lateral rectus muscles was done for the exotropia and a bilateral 10-mm. recession of the inferior obliques for the vertical deviations.

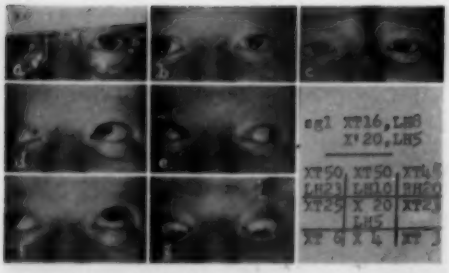

Fig. 8A (Urist). Case 7, preoperative examination.

a. Eyes straight for near; no fusion obtainable.

b. Twenty degrees of right exotropia for distance. (Note only 16 prism diopters of exotropia found with cover measurements.)

c. Near-point of convergence is 50 mm.

d. Marked elevation of the left eye in adduction.

e. Marked elevation of the right eye in adduction.

f. Thirty-five degrees of right exotropia looking up.

g. Eyes straight looking down.

Postoperative examination on November 8, 1944, two months after surgery (fig. 8B) revealed about 10 degrees of exotropia for distance, although the eyes could be straight for distance with esophoria measurements, straight eyes for near with stereopsis, and a convergence near-point of 30 mm. Looking up, the exotropia was improved although 20 to 25 degrees still remained.

The cover measurements looking up were XT 20 prism diopters instead of the XT 50 prism diopters found before surgery.

The eyes remained straight looking down. The bilateral elevation in adduction was not improved following the recession of the obliques and the vertical cover measurements remained the same.

Examination on November 26, 1951, two years after surgery (fig. 9) revealed straight eyes for distance and near with stereopsis. The eyes were straight looking down with 20 to 25 degrees of exotropia looking up. The bilateral elevation in adduction was still marked but the vertical cover measurements were much smaller, having improved from a left hypertropia of 24 prism diopters to a left hypertropia of 14 prism diopters and from a right hypertropia of 20 prism diopters to a right hypertropia of six prism diopters.

The improvements in the vertical cover measurements were most likely due to the stereopsis and good fusion amplitude which developed over a period of two years following the correction of the exotropia for distance.

Contrary to what might be expected following the bilateral recession of the inferior oblique muscles, there was no visible improvement in the elevation in adduction. Perhaps in these cases having such a large exotropia looking up and large vertical deviations, a bilateral 10-mm. recession of the lateral rectus muscles would be the indicated procedure. Certainly the 8.5-mm. recession caused no limitation in abduction. The larger recessions may correct the exotropia looking up which would most likely eliminate the dissociated vertical deviations.

Discussion of Table 1

My results with surgery, presented in Table 1 (all of these cases have been followed from one to seven years after sur-

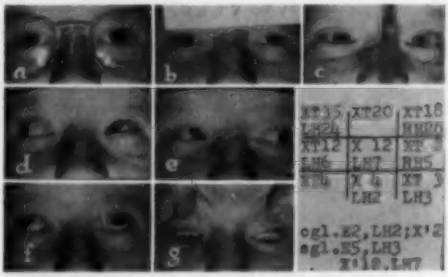

Fig. 8B (Urist). Case 7, postoperative examination, two months after a bilateral 8.5-mm. recession of the lateral rectus muscles and a bilateral 10-mm. recession of the inferior oblique muscles.

a. Five degrees of left exotropia for near with glasses (can be straight). Patient has stereopsis at 0.

b. Ten degrees of left exotropia for distance; can also be straight.

c. Near-point of convergence is 30 mm.

d. Marked elevation of the left eye in adduction.

e. Elevation of the right eye in adduction.

f. Twenty degrees of right exotropia looking up.

g. Eyes straight looking down.

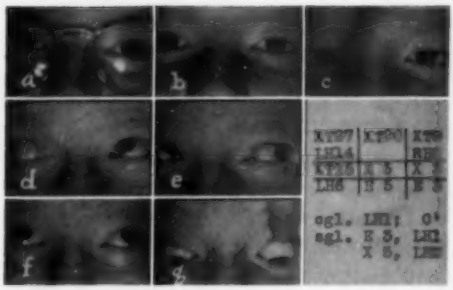

Fig. 9 (Urist). Case 7, postoperative examination, two years after surgery.

a. Eyes straight for distance and near with glasses; third-grade fusion.

b. Eyes straight for distance and near without glasses.

c. Near-point of convergence is 60 mm.

d. Elevation of the left eye in adduction.

e. Elevation of the right eye in adduction.

f. Twenty degrees of right exotropia looking up.

g. Eyes straight looking down.

gery), confirm the results of Scobee which show that the bilateral recession of the lateral rectus muscles gave a functional cure in the majority of cases. Scobee[1] was so enthusiastic about his results that he stated:

"The results of surgical therapy have been so encouraging that resident physicians nearing completion of their service have expressed the fervent hope that their first patient in private practice will have intermittent exotropia."

In our series the functional cures were consistently obtained with the bilateral recession of the lateral rectus muscles in those cases that had an exotropia for distance and upward gaze and straight eyes for near and looking down. The ability to hold the eyes straight for near and looking down maintained vision and developed fusion amplitude

TABLE 1

RESULTS OF SURGERY IN 54 OF MY CASES AND IN SCOBEE'S 25 CASES

Type of Surgery	No. of Cases	Functional Cure	Cosmetic Result Only	Under-Correction	Over-Correction
Bilateral recession of the lateral rectus muscles	22	20	0	1	1
Scobee 1949	25	21	2	2	0
Bilateral tenotomy of the lateral rectus muscles	6	2	1	3	0
Bilateral recession of the lateral rectus muscles and unilateral resection of the medial rectus muscle	1	1	0	0	0
Bilateral tenotomy of the lateral rectus muscles and unilateral resection of the medial rectus muscle	6	1	3	1	1
Unilateral resection of a medial rectus muscle and recession of a lateral rectus muscle	9	1	0	7	1
Unilateral tenotomy of a lateral rectus muscle and resection of a medial rectus muscle	3	0	0	2	1
Unilateral recession of a lateral rectus muscle	3	1	0	2	0
Unilateral resection of a medial rectus muscle	1	0	0	1	0
Bilateral resection of the medial rectus muscles	3	0	0	2	1

in these cases thus insuring a functional cure postoperatively.

Of the 22 cases that had bilateral recession of the lateral rectus muscles the distribution of the amount of surgery in the cases was: five mm. in one case, six mm. in two cases, seven mm. in 10 cases, eight mm. in seven cases, 8.5 mm. in one case, and 9.5 mm. in one case.

The one case that was undercorrected had the bilateral five-mm. recession which I believe was insufficient to correct the large exotropia present on upward gaze (25 degrees) and for distance. This patient was followed for three years and had to be reoperated.

The overcorrection was in the case that had the bilateral 9.5-mm. recession. In this case the exotropia looking up and for distance before surgery was small (15 degrees) and the bilateral 9.5-mm. recession overcorrected it. Even with such a large recession very little limitation in abduction was present postoperatively.

As may be seen, the bilateral simple tenotomy gave no overcorrection, but, in the majority of cases, an undercorrection. The resection of the medical rectus muscles alone as a primary procedure gave consistently poor results. Unilateral resection of a medical rectus and recession of a lateral rectus muscle usually produced an undercorrection.

Comment

Cases of exotropia with bilateral elevation in adduction have apparent overaction of the lateral rectus muscles. The overaction is manifested by the exotropia for distance and upward gaze being greater than for near and downward gaze.

For surgery the cases can be divided into two types:

1. Those with no underaction of the medial recti, manifested by straight eyes for near and downward gaze.

2. Cases with underaction of the medial recti manifested by an exotropia for near and downward gaze which is less however than that for distance and upward gaze.

The indicated surgery for the cases with only an exotropia for distance and upward gaze (type 1) is a bilateral recession of the lateral rectus muscles.

Scobee[2] advocated a bilateral recession of the lateral rectus muscles to the equator. It was unfortunate that he did not give the exact amount of recession that was done.

According to Marshall, as quoted by Spaeth,[3] the distances along the circumference of the globe can vary as follows:

The distance between the limbus and the ora serrata varies from six to eight mm. in normal eyes and from nine to 10 mm. in highly myopic eyes. The distance between the ora serrata and the equator varies from six to eight mm. This means that the distance from the limbus to the equator can vary from 12 to 16 mm. in normal eyes and 15 to 18 mm. in highly myopic eyes. The insertion of the lateral rectus muscles is 6.9 mm. from the limbus (Wolff[4]).

With these figures in mind, a recession can vary from five to 11 mm. before the muscle is placed to the equator, depending on the eye. It is very difficult to determine the location of the equator at surgery. Many surgeons use the average figure of 13 mm. as the distance of the equator from the limbus and will not do more than a six-mm. recession of a lateral rectus muscle. A recession of more than six mm. has not given poor results in my practice.

In my experience, the amount of bilateral recession may vary from five mm. for a small amount of exotropia on upward gaze, to nine mm. for a large exotropia on upward gaze. Case 5 had a 10-mm. recession of one lateral rectus muscle with no limitation in abduction. Case 7 had a bilateral 8.5-mm. recession with a large exotropia remaining on upward gaze and no limitation in abduction. Perhaps in this case a bilateral 10-mm. recession would have corrected the exotropia on upward gaze.

As may be seen in the discussion of Table 1, most of my cases had a bilateral seven- or eight-mm. recession of the lateral rectus muscles with excellent results. In no case was there significant limitation in abduction following surgery.

I have found that there is a definite anatomic basis for a recession of even 10 mm. in indicated cases, and without crippling the action of the lateral rectus muscle. A study of the illustration by Adler[5] will make the following discussion much clearer.

The anatomic equator of the eyeball is a line encircling the globe midway between the anterior and posterior poles. The diameter at the equator would be perpendicular to the optic axis. The visual axis is about five degrees medial to the optic axis (angle kappa). The orbital axis is about 20 degree lateral to the optic axis (Whitnall).

The rectus muscles originate from the apex of the orbit and insert onto the eyeball which is approximately in the center of the exit of the orbit. Because of this, the rectus muscles would have their arc of contact with the globe and rotating effect relative to the orbital axis and a functional equator and not to the optic axis and anatomic equator. This would be a line encircling the globe dividing the eyeball in half along the orbital axis. The diameter at the functional equator would then be perpendicular to the orbital axis.

Since the orbital axis is 20 degrees lateral to the optic axis, the functional equator would be about 20 degrees posterior, laterally, and 20 degrees anterior, medially, to the anatomic equator, and since five degrees equal about one mm. on the circumference of the globe, the functional equator would be about four mm. more posterior, laterally, and about four mm. more anterior, medially, than the anatomic equator.

Therefore, the lateral rectus can be recessed about four mm. back of the anatomic equator to the functional equator before it loses its arc of contact. The medial rectus should not be recessed to the anatomic equator as advocated by Scobee[7] because it would lose its arc of contact about four mm. anterior to it.

The bilateral simple tenotomy (no check ligaments cut) of the lateral rectus muscles was abandoned because it usually gave an undercorrection.

For cases with an exotropia for near and looking down as well as for distance and looking up (type 2), a unilateral (Case 3) or bilateral (Case 5) resection of the medial rectus muscles is indicated along with the bilateral recession of the lateral rectus muscles. If the resection of the medial rectus is not done, the exotropia looking down may not be corrected, as in Case 2.

The bilateral resection of the medial rectus muscles for cases in this group as a primary procedure, especially for the type-1 cases, is not the best choice of surgery (Table 1). The overaction of the lateral rectus muscles is not corrected and overaction of the medial rectus muscles is produced.

Case 6 shows the effect of a single resection of the medial rectus. It produced an esotropia looking down where the eyes were straight before surgery. The overaction of the lateral recti with the large exotropia looking up still remained.

Good results in the central field may be found after resection of the medial recti in some of these cases, but, when the straight-up and straight-down positions are examined, it will be found that the straight eyes in the central field was brought about by a balance between two overactions, that is, the original overaction of the lateral recti seen by the exotropia which remained on looking up, and the surgically induced overaction of the medial recti as shown by the esotropia on looking down (fig. 7B).

Cases in this group with marked elevation in adduction have been diagnosed as primary superior rectus pareses with overaction of the inferior obliques, and bilateral recessions of the inferior oblique muscles have been

done to correct the overactions.

In these cases (three cases in our series) which had bilateral recessions of the inferior oblique muscles and insufficient lateral muscle surgery to correct the exotropia for distance and especially looking up, the bilateral elevation in adduction remained and the vertical deviations were not improved (Case 7).

On the other hand, when sufficient surgery to the horizontal muscles was done to correct the exotropia in all positions of gaze, the vertical deviations became much improved and in many cases completely disappeared (Case 5). In order for the dissociated vertical deviations to be alleviated, the exotropia had not only to be corrected for the central fields but also for the straight up and straight down positions of gaze.

Summary

1. Cases of exotropia with bilateral elevation in adduction have apparent overaction of the lateral rectus muscles (divergence excess) manifested by a greater exotropia for distance and upward gaze than for near and downward gaze.

2. A bilateral recession of the lateral rectus muscles is indicated for those cases in which the eyes are straight for near and looking down. A unilateral, and, more rarely a bilateral, resection of a medial rectus along with the bilateral recession is indicated for those cases that also have an exotropia for near and looking down.

3. The bilateral resection of the medial rectus muscles is contraindicated as a primary procedure for the cases that have straight eyes for near and looking down (most cases in this group) because it produces overaction of the medial rectus muscles without correcting the primary defect which is overaction of the lateral rectus muscles and may result in an esotropia on looking down.

4. The bilateral recession of the inferior obliques alone will not correct the bilateral elevation in adduction and the vertical deviations since they are secondary and depend on the presence of the exotropia. Correction of the exotropria in all positions of gaze will improve the vertical deviations so that the inferior obliques may not have to be recessed.

5. Anatomic evidence indicates that the medial and lateral rectus muscles have their arc of contact relative to a functional equator on the globe. This allows more recession of the lateral rectus muscles and less recession of the medial rectus muscles relative to the anatomic equator.

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INTERMITTENT AND ACCOMMODATIVE ESOTROPIA*

A STUDY OF 74 CASES

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This report represents an analysis of 74 cases of intermittent and accommodative esotropia. We have used the term intermittent esotropia for those cases which show both esophoria and esotropia. They may have an esophoria for distance and an esotropia for near, or they may be intermittent both for distance and near. There were 30 cases of this type. In contrast, the average case of accommodative esotropia is a uniocular obligatory squint which is converted by corrective lenses to intermittent esotropia, to esophoria, or to orthophoria.

With few exceptions the cases of intermittent esotropia are accommodative and would seem to be an intermediate group between the true accommodative esotropias and the esophorias. Because these two types of squint are so closely related and have almost interchangeable features, we have grouped them together.

In excluding patients whose strabismus is not significantly corrected by the wearing of glasses, and those requiring surgical correction for improvement, we have focused our attention on patients who show both esophoria and esotropia in the hope that we might obtain some information about the pathogenesis of esotropia in general.

At the first examination of a patient with a constant esotropia, the ophthalmologist cannot foretell whether this case may be corrected by nonsurgical methods alone. We have attempted to find some clues which would enable us to predict the ultimate success of nonsurgical treatment in any particular case. With this in mind, a survey revealed the following factors to be the most significant:

1. Age of onset
2. Visual acuity
3. Refractive error
4. Near-point of convergence
5. Fusional status
6. Progress of the cases

While the patients displayed a great deal of uniformity, many showed wide variations from the group average in one or more of the above-mentioned factors.

AGE OF ONSET

In 77 percent of the cases the parents reported that the strabismus began between two and six years of age. In 14 percent the onset was before the age of two, and in nine percent the parents noted the squint after the age of six years (table 1).

VISUAL ACUITY

While most of the cases were uniocular, 15 percent were classified as so-called alter-

TABLE 1

AGE OF ONSET

Age (years)	Number of Cases
Birth to 1 year	3
1 to 2	7
2 to 3	11
3 to 4	16
4 to 5	13
5 to 6	10
Over 6	5

* From the Department of Motor Anomalies, New York Eye and Ear Infirmary. Presented in part before the Section of Ophthalmology, New York Academy of Medicine, April 20, 1953.

TABLE 2

VISUAL ACUITY

Both eyes equal to 20/20 or 20/30	36
One line difference between eyes (20/20 in one eye and 20/30 in other)	21
Two lines difference between eyes (20/20 and 20/40)	9
Difference between eyes three lines or more	5

nators. Contrary to findings in most patients with obligatory uniocular strabismus, very few in this group showed any marked degree of amblyopia (table 2). In 80 percent of the cases, the vision was equal (20/20 or 20/30) in both eyes or there was but one line of difference on the Snellen chart; 13 percent had two lines of difference; and another seven percent showed three or more lines of difference.

The visual acuity in the amblyopic eyes of most of these patients improved as the eyes became straight. In most cases this improvement occurred without the benefit of orthoptic training, whereas others required patching of the fixing eye. Only three patients retained an amblyopia of 20/50 or less despite improvement in the angle of squint.

REFRACTIVE ERROR

Under atropine all of the patients, with but one exception, had hyperopia greater than one diopter (table 3); 23 percent of them had astigmatism of one diopter or more and were included in the total statistics by using their spherical equivalents. Of these cases, 48.7 percent had hyperopia between 3.25 and 5.00 diopters, while 29.7 percent showed a greater, and 21.6 percent a lesser refractive error. Three patients had anisometropia greater than 2.75 diopters. There was no relationship between the amount of hyperopia and the degree of strabismus.

TABLE 3

REFRACTIVE ERROR*

Refractive Error	Number of Cases
0 to +1.00	1
+1.25 to +2.00	9
+2.25 to +3.00	12
	22 (29.7%)
+3.25 to +4.00	20
+4.25 to +5.00	16
	36 (48.7%)
Other +5.00	16 (21.6%)

* Cases of astigmatism were included by using their spherical equivalents.

NEAR-POINT OF CONVERGENCE

It is interesting that 42 percent of the patients had a near-point of convergence more remote than 29 mm. It would seem that this is a large percentage in a group of cases with convergent strabismus. Nevertheless, the near-point had no relation to the progress of the condition and patients with a more remote near-point did as well as those whose near-point was unlimited.

FUSIONAL STATUS

As one would expect, the patients in this group showed a very high degree of fusion as compared to other types of convergent strabismus (table 4). In every case where the retinal correspondence was determined it was normal. When possible, the major amblyoscope was used and 47.3 percent showed first, second, and third-grade fusion. Another 24 percent reported fusion with the Worth four-dot test although they were not examined with the amblyoscope; 9.4 percent appeared to have intermittent fusion; and only nine percent failed to show evidence of fusion by the tests used.

TABLE 4

FUSIONAL STATUS

Amount of Fusion	Number of Cases	Percentage
First, second and third grade	35	47.3
First and second or second and third grade	8	10.9
Fusion with Worth 4-dot test	17	23
Intermittent fusion	7	9.4
No fusion	7	9.4

Progress of the cases

The degree of strabismus varied a great deal among the cases. The patient with the smallest amount of strabismus measured orthophoria for distance and an intermittent esotropia of eight prism diopters for near, while the one with the largest amount of squint had an esotropia of 30 prism diopters for distance and an esotropia of 55 prism diopters for near. The majority, 45 cases, measured less than 35 prism diopters of esotropia for near with the screen and prism test.

In accommodative strabismus one would expect the amount of strabismus to be greater for near than for distance. This was true in all but eight patients. The most interesting of these exceptions was a patient with an esotropia of 25 prism diopters for distance and orthophoria for near. After wearing full hyperopic correction for two years and three months, this patient measured four prism diopters of esophoria for distance and orthophoria for near.

Taken as a group, these patients responded promptly and well to their full corrections regardless of the amount of squint. In some cases the strabismus was converted to esophoria or orthophoria within two to three months, while in other cases it took over a year before the strabismus was fully corrected. Since the response to the correction of refractive errors is so uniform only three examples will be given to show the typical course in the average case.

Case reports

Case 1

A. S., aged four years, was first seen in May, 1950. At this time she had an esotropia of 20 prism diopters for distance and 50 prism diopters for near without correction. After wearing a +1.25 D. sph. ⌒ +1.0D. cyl. ax. 90°, O.U., for six months, the strabismus was reduced to orthophoria for distance and a variable intermittent esotropia of about four prism diopters for near. Sixteen months later, with hyperopic correction, she was orthophoric for both distance and near.

Case 2

M. C., aged four years, was first examined in May, 1949. He had an esotropia measuring 15 to 20 prism diopters for distance and near. After wearing his full correction, +4.5D. sph., O.U., for three months he was orthophoric with his glasses.

Case 3

J. M. was first seen at the age of five years. Her strabismus measured 30 diopters of esotropia for distance and near. After wearing: R.E., +1.5D. sph. ⌒ +1.0D. cyl. ax. 90°; L.E., +2.0D. sph., for 11 months, her deviation measured six prism diopters of esophoria for distance and near with correction.

Concurrent with the decrease in the degree of squint, it was noted that whatever initial amblyopia was present frequently disappeared and many patients showed evidence of an improvement in fusion. Improvement occurred in many patients without orthoptics or even patching of the fixing eye. Only three cases were felt to warrant orthoptic training, indicating that the great majority of these cases need only removal of the accommodative element in order to improve. Corrective surgery was not performed on any of these patients.

In seven patients, the strabismus for distance improved to orthophoria but a small amount of esotropia remained for near even after the patients wore their full corrections for some length of time. Bifocals were prescribed for these children, with beneficial results. The bifocal prescribed is that amount with which the patient reads 20/30 on the Lebensohn chart. It could be as much as an add of +5.00 diopters.

One child who originally measured 20 prism diopters of esotropia for both distance and near, wore a correction of +3.00, O.U., for five months. He then measured ortho-

phoria for distance but still retained an esotropia of 12 prism diopters for near. He was advised to wear bifocals and his esotropia for near was improved to esophoria of four prism diopters.

We believe that bifocals should be used only by patients whose glasses fully correct the esotropia for distance but who still have a small residual constant or intermittent esotropia for near. The bifocal should be strong enough to correct the strabismus for near. The diameter of the segment must be large enough to be situated at the border of the pupil. The strength of the segment can gradually be diminished over a period of several years. If the child is old enough, the orthoptist can treat him in an attempt to dissociate the faulty accommodation-convergence linkage.

Conclusion

From this study, it would seem that a case of convergent strabismus with the following characteristics has a reasonable chance of correction by nonsurgical means:

1. Onset between four and six years of age
2. Relatively equal vision in both eyes
3. Hyperopia under atropine between three and five diopters
4. Evidence of a well-functioning fusion faculty.

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We wish to acknowledge the help of Miss Dorothy Parkhill and Miss Leta Counihan of the Orthoptics Department in preparing this study.

TOXOPLASMOSIS*

A report of the literature and clinical studies based on five cases

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Introduction

Since the early descriptions of toxoplasmosis, many clinical and investigative reports have appeared concerning the disease. Practically all of these have given cognizance to the presence of ophthalmic pathologic conditions. It is the purpose of this paper primarily to review toxoplasmosis from the ophthalmic standpoint, to review the literature,[1-23] and to present five cases which had previously been undiagnosed.

Discovery

Nicolle and Manceaux, in 1908, demonstrated the protozoan parasite, Toxoplasma, in the North African Gondi. Splendor, working independently, in the same year, described the organism in the Brazilian rabbit.

Morphology

The organisms[2] are crescentric, oval, or pyriform in shape, and vary in size from two to seven microns in length and from two to four microns in width. They may be pointed at the ends or one end may be blunted. The nuclear chromatin (there is no nuclear membrane) stains deep purple and the cytoplasm, pale blue, with Wright's stain.[14] In fixed tissues, the organisms appear smaller and rounder and may exhibit features in common with ultramicroscopic viruses. The organisms may occur in clusters forming cysts or pseudocysts.

Reproduction

Multiplication occurs by binary fission in the longitudinal axis and is possible only

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within living cells. Efforts to culture the organisms on media have failed as they are obligate intracellular parasites.[4] Cyst formation[21] is produced by continued multiplication within an individual cell.

As the cell continues to fill with organisms and becomes distended, its nucleus is extruded, leaving a cluster of organisms, at times in the hundreds, surrounded by a cell membrane.

After the passage through many successive hosts the pathogenicity of the organism increases. Following this only a few subsequent generations of this increased highly virulent strain is necessary to make the host cell completely disintegrate. The same increase in pathogenicity, with resultant cell disintegration, may occur spontaneously after only a few successive passages, in a less virulent type.

Animal inoculations

Prior to the crystallization of reliable serologic diagnosis, a considerable number of animal studies[4] were performed; further investigation was founded upon them. The disease was found to be fatal in mice, guinea pigs, rabbits, and chickens. The Rhesus monkey, however, survived inoculation.

In the mouse, which is particularly susceptible, the brain was involved despite the mode of inoculation. With intracerebral inoculation, the organisms were distributed by the cerebrospinal circulation and the lesions situated under the meninges along the canal. Peripheral inoculation resulted in a vascular distribution, the Toxoplasma penetrating and infecting the cells of the vessels and the perivascular nervous tissue. The lungs were usually next involved, with the parenchymal organs being involved only slightly.

Rabbits survived intracutaneous inoculations prior to spontaneous increase in pathogenicity. Following the change, the lesions showed greater necrosis and hemorrhage with death following in eight to 12 days. The monkeys responded to intracerebral inoculation with only a febrile rise. Recovery produced immunity to reinfection with a similar strain or one of increased virulence.

An in vitro mixture of organisms with immune monkey serum protected mice and rabbits. The organisms, however, retained their infectivity, as was demonstrated when the immune serum was removed from the mixture; the remaining organisms produced fatal disease upon inoculation. Rabbits which had recovered from weak strains of subcutaneous inoculation presented an immunity to reinfection and also to strains of increased virulence.

In 1939, Wolff, Cowan, and Paige[6] reported a case of infantile encephalitis with chorioretinitis in which the patient died at 31 days of age. Toxoplasmalike parasites were found in the brain. Prior to this, five cases of encephalitis, three exhibiting chorioretinitis, had been described and in each a parasite morphologically identical with Toxoplasma had been observed.

Using a saline emulsion of brain tissue, Wolff, Cowan, and Paige successively inoculated rabbits, mice, and rats. The strain was repeatedly passed to subsequent hosts. The animals become ill and died or were killed in 18 to 40 days.

The central nervous-system lesions resembling those found in the human were described. Morphologically, similar protozoa to those seen in the human were found, and further transmission was successful. Koch's postulates had been fulfilled.

This was the first case of human toxoplasbosis transferred to animals. Subsequently Sabin[8] reported animal and human toxoplasmosis to be biologically identical and that immunity against one would produce immunity to the other.

Serologic examinations

Prior to the serologic tests,[9] diagnosis would only be made at autopsy or by animal passage which became significant only after the patients had recovered. The rabbit neutralization test is dependent upon the ability of human serum to modify the lesions pro-

duced by intracutaneous inoculations of a rabbit. The reaction depends upon a specific antibody which is heat stable and a nonspecific factor related to complement which is found in normal human serum and is heat labile.

The dye test which is more quantitative is performed as follows:

The suspected serum is serially diluted. To this is added a mixture of Toxoplasma organisms and normal human serum containing the accessory factor. It is then incubated for one hour at 37°C. Portions of each tube are then mixed with alkaline methylene blue, pH 11.0.

The percent of extracellular Toxoplasma organisms which will maintain clear cytoplasm is determined. When 50 percent of the organisms no longer remain clear, the end-point of the titration has been reached. Normal human serum rarely gives a positive test in greater dilution than 1:64.[11]

During the first few weeks of infection, the dilution might not exceed 1:64. Within 10 to 21 days following infection, the dye test becomes positive. However, the complement-fixing antibodies may not be demonstrated for one month following infection. An increasing complement-fixating reaction in the presence of a positive dye test may be the only indication of an active infection.

Following toxoplasma infections, antibodies persist in the serum for as long as six years—as noted by Sabin.[10] In 17 mothers giving birth to children with toxoplasmosis, all mothers had a positive complement fixation test in dilutions of 1:4 and 1:64, the highest titers being present in the first two years. A toxoplasmic antigen was developed for intradermal testing.[23]

In 1949,[19] a report describing the results of tests on 142 people unsuspected of having toxoplasmosis was published. Of the following age groups, the percentage of people giving a positive test was: 0-4, 0 percent; 5-9, 5 percent; 10-19, 14 percent; 20-29, 20 percent; 30-39, 50 percent; 40-49, 65 percent.

Antibody was present in the cases with positive skin tests. No agent having antigenic capacity similar to toxoplasmosis has been demonstrated. A positive skin test, however, gives no relation to the antibody level while negative skin tests have been encountered in the presence of antibody. That placentally transmitted toxoplasmic antibodies exist in normal children born to mothers who have had toxoplasmosis has been demonstrated.[18]

The children of three mothers who had given birth to previous children infected with toxoplasmosis were studied with both the dye test and the complement-fixation tests. They remained positive with undiminshed titers for the first six weeks. At the end of four and one-half months, there was a marked diminution of antibody. Nine months following birth, a dye-neutralization antibody was found in stronger concentration than the complement-fixating antibodies but greatly diminished in concentration.

Initial tests during the first six weeks of life are of little value unless subsequent examinations are made after four months in children born to mothers having positive serologic tests for toxoplasmosis.

Clinical classification of toxoplasmosis

The disease may occur during neonatal life, infancy, and childhood,[18] and also appears in acute[16, 17] form in adults. Probably the most baffling form, however, is that which occurs, unrecognized, in pregnant women and becomes tragically evident in their offspring.

The neonatal form is usually characterized by encephalitis, rash, jaundice, hepatosplenomegaly, hydrocephalus, and chorioretinitis. During infancy and childhood, the symptom complex consists of convulsions, microcephalus, psychomotor retardations, chorioretinitis, and cerebral calcification. Together these cases are grouped as congenital toxoplasmosis and, as such, the most common symptoms are chorioretinitis, cerebral calcification, hydrocephalus or microcephalus, and psychomotor disturbance.

The disease may be full blown at birth, if

transmitted early in pregnancy. If, however, it is transmitted late in pregnancy, the disease may not develop until a few weeks following birth.

The adult form of the disease starts with a prodromal stage consisting of malaise and weakness for several days. This is followed by sudden onset of the acute stage, marked by chills and fever. A maculopapular rash develops and the cutaneous lesions have been shown to contain the Toxoplasma organism. Pneumonitis with death usually due to interference with respiratory function may follow. An acute encephalitis and myocarditis have been observed. Cerebral calcification and chorioretinitis are usually lacking.

Pathology

The lesions of toxoplasmosis are generally confined to the neural tissue but may be disseminated in any of the parenchymal organs and in the skin. Pathologic lesions containing organisms have been described in the stomach, small intestine, large intestine, pancreas, lungs, liver, kidney, hypophysis, thyroid, adrenal, and skeletal muscles.[10]

Microscopic pathologic sections reveal necrotic tissue surrounded by a few polymorphonuclear leukocytes. A greater concentration of lymphocytes and plasma cells, eosinophils and fat-laden phagocytes is observed. Scattered areas of calcification may be evident. In the brain, perivascular cuffing, in addition to the focal necrosis, is more evident.

Pathologic findings in the retina, as described by Wolff, Cowan, and Paige,[6] are:

"The retina showed edema of all its layers and distortion of the opticum, ganglionic, and inner molecular layers. The capillaries in this zone had undergone endothelial hyperplasia. Many nerve cells in the ganglionic layer were partially degenerated.

"The layers of rods and cones were markedly necrotic. The pigmented layer was partially disrupted and destruction of its cells gave rise to much extracellular pigment.

"The choroid was congested, showed capillary hyperplasia, and was infiltrated by plasma cells, lymphocytes, and eosinophils. The leptomeningeal sheath of the optic nerve showed a similar mild infiltration."

Ophthalmic considerations

The problem of toxoplasmosis in ophthalmology has been adequately described in many previous reports and is well demonstrated by the cases reported in this paper.

Encephalitis with chorioretinitis appears to be the most common finding in the disease at present. Chorioretinitis is of extreme value in making the diagnosis when there is congenital cerebral damage due to toxoplasmosis. If not present at birth, the fundus lesions may develop within a few weeks. The macula is most frequently involved. This was thought to be of particular importance in the early descriptions but lesions have been observed in all quadrants of the retina.

The findings of a previous iritis, as evidenced by posterior synechia, uveal pigment on the lens, and iris atrophy, as in Case 2, would indicate that the lesions may occur anywhere within the uvea. The lesions may be bilateral or unilateral.

In considering the diagnosis of the congenital type, the serologic pattern becomes important. A passive transfer of antibody can lead to an erroneous diagnosis if the examinations are not repeated after six weeks of age, particularly if the mother had given birth to an infected child within the past two years. Although differential diagnosis may not be considered, the usually accepted causes of chorioretinitis in infants must be investigated.

It is noteworthy that two of the five cases herein reported were in premature infants and a third was that of a baby of seven and one half months' gestation but weighing 5 lb. 5 oz. One hundred cases of retrolental fibroplasia have been found negative for toxoplasmosis.[15]

Koch[18] reports the following ophthalmic abnormalities with toxoplasmosis: persistent pupillary membrane, narrowing of the pal-

pebral fissure, microcornea, posterior cortical cataracts, posterior lenticonus, severe anisometropia, optic atrophy, papilledema, nystagmus, muscle weakness. My Case 1 exhibited a persistent hyaloid artery and Bergmeister papillae.

Many of these abnormalities have been in eyes in which chorioretinitis was also present.

Does toxoplasmosis have an effect, perhaps similar to that of German measles, upon the developing embryo which results in these abnormalities?

Vail has described six cases of progressive loss of vision due to chorioretinitis in which a diagnosis of toxoplasmosis was made. Loss of visual acuity was the only symptom.

Two of the children reported in this paper (Cases 3 and 4) were found to be normal in every respect except for ocular involvement.

Wilder[22] reports organisms resembling Toxoplasma in 41 enucleated eyes in which a diagnosis of uveitis had been made. Sabin,[18] however, reports that, without corroborating serologic studies, the pathologic diagnosis cannot be relied upon, since other protozoan bodies may resemble Toxoplasma in tissue sections.

Treatment

The treatment of toxoplasmosis is without much success. Sulfadiazine and Sulfamerazine have been shown to control acute toxoplasmosis, probably by suppressing proliferation of organisms. Promine and Endochin have been effective in acute experimental toxoplasmosis. The newer antibiotics are without effect.[24]

Case reports

Case 1

D. T., aged 12 days, was admitted because of enlargement of the head.

Spinal puncture showed clear yellow fluid; cell count: 124; protein: 280 mg. percent; sugar: 45 mg. percent; chloride: 630 mg. percent. Yellow fluid was also recovered from ventricular taps.

Neutralizing titer for toxoplasmosis of the child's serum was positive in dilution of 1:1,024. The mother's serum was positive in dilution of 1:2,048. Two months later a titer of 1:4,096 was obtained for the child. The mother's titer was unchanged. Linear intracranial calcification was noted on X-ray examination. The electro-encephalogram was normal.

Right eye. There was a fringelike remnant of pupillary membrane extending around the entire pupil and a persistent hyaloid artery with Bergmeister's papillae.

Left eye. The vitreous contained cellular material. The nervehead, as in the other eye, was pale and irregular. Superiorly to the nervehead, a large grayish-white area was seen. The entire area was probably an active chorioretinitis which did not present pigmentation at that time.

The head continued to enlarge. The child developed intermittent generalized convulsions and death followed. Autopsy revealed toxoplasmosis of the brain (figs. 1 and 2), hydrocephalus, edema of the lungs, acute myocarditis possibly due to toxoplasmosis. The eyes could not be obtained.

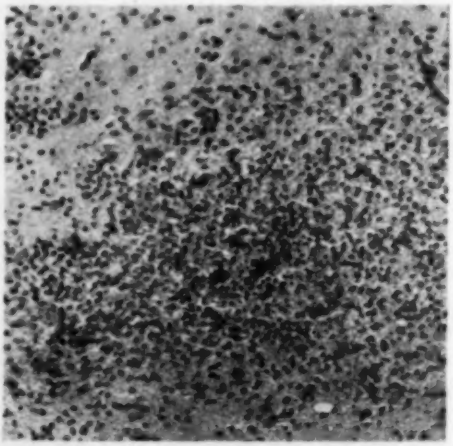

Fig. 1 (Masters). Focus of necrosis in cerebrum in toxoplasmosis. There are numerous neutrophils and microglia, some of which are of the glitter-cell type (hematoxylin-eosin, ×100).

CASE 2

W. W., aged two months, was admitted because of hydrocephalus and narrowing of the right palpebral fissure. Spinal puncture showed: clear yellow fluid; lymphocytes: 10; Pandy: 4+; chloride: 438 mg. percent; a clear light-yellow fluid was obtained from ventricular punctures.

Complement-fixating titers for toxoplasmosis revealed the child's serum to be positive in dilution of 1:32. The mother's serum was positive in dilution of 1:64. The dye test for toxoplasmosis was positive in dilution of 1:618 for mother and child.

Diffuse mottled intracranial calcification was noted on X-ray examination. Ventriculogram revealed diffuse internal hydrocephalus. A craniotomy was performed to relieve pressure and biopsy material obtained.

Right eye. The pupil was small and irregular and did not react to light, and did not dilate with mydriatics. There were dense posterior synechias with pigment deposits on the lens. The iris was atrophic. The media contained vitreous floaters. The macula was clear. Several disc diameters above the nervehead there was a dark-grayish area which was surrounded by flecks of black pigment.

Pathologic report of the specimen obtained revealed lesions due to toxoplasmosis. The child was discharged unimproved and subsequently died.

CASE 3

J. G., aged one and one-half years, was born after seven and one half months' gestation with weight at birth 5 lb. 5 oz. The mother first noticed that the right eye turned in at six months of age. A large, white, lobulated mass, having six diopters of elevation, filled the right macula. The complement-fixation test for toxoplasmosis was positive. The dye test for toxoplasmosis was positive in dilution of 1:4,096. The mother's serum was positive in dilution of 1:16 for the dye test.

Spinal puncture showed fluid clear; protein: 10 mg. percent; sugar: 50 mg. percent; chloride: 667 mg. percent. X-ray examination of the skull was normal.

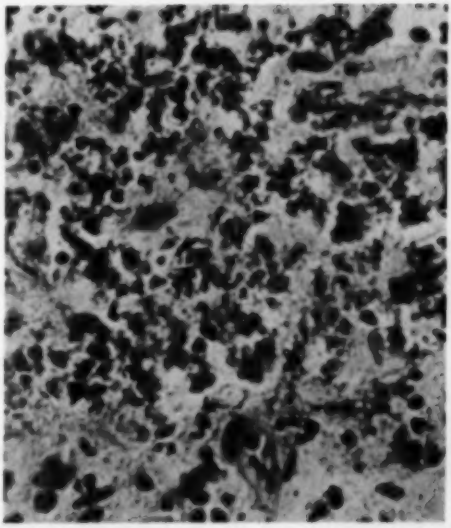

Fig. 2 (Masters). Toxoplasmosis organisms in a necrotic focus in cerebral cortex. These are to be distinguished from the larger granules which represent fragments derived from necrotic nuclei. (Hematoxylin-eosin, ×450.)

Six months after the initial lesion in the right eye was noted, the following changes were seen:

The yellowish elevated lesion in the macula had flattened considerably and appeared as a circumscribed dirty gray area which completely filled the macula. At the periphery, blood vessels were seen approaching the lesion. Almost adjacent to the macula but slightly inferiorly and temporally, there was a round area, one disc diameter in size, of pigmented chorioretinal degeneration.

CASE 4

N. M., aged six and one-half months, was born after six and one-half months' gestation and weighed 2 lb. 10 oz. at birth. The patient was admitted following the discovery of an area of chorioretinal degeneration in the extreme periphery of the upper temporal quadrant, O.S. No macula lesions were present.

Spinal puncture showed clear fluid; cell count: 40 lymphocytes; sugar: 50 mg. percent; protein: 20 mg. percent. Wassermann, negative.

Toxoplasmosis studies revealed the dye test to be positive in dilution of 1:1,224; the complement-fixating titer was positive in dilution of 1:8. Examination of the mother's serum revealed the dye test to be positive in dilution of 1:2,448 and the complement-fixating titer to be positive in dilution of 1:16. X-ray examination of the skull failed to reveal any intracranial calcifications.

Case 5

A. R., aged eight months, had a birth weight of three pounds. Gestation period was seven months. The patient was admitted for enlargement of the head.

Spinal puncture showed clear yellow fluid; cell count: 24 lymphocytes; glucose: 51 mg. percent; chlorides: 655 mg. percent.

Neutralizing titer for toxoplasmosis of the child's serum was positive in dilution of 1:1,024. The mother's serum was positive for toxoplasmosis by the complement-fixation test. Diffuse linear intracranial calcification and dilated ventricles were observed on X-ray examination. Examination of the eyes revealed bilateral optic atrophy.

Conclusions

1. Five previously undiagnosed cases of human toxoplasmosis are reported.

2. Ophthalmic lesions in children having toxoplasmosis may occur as part of a general syndrome or may be the only demonstrable pathologic process.

3. Toxoplasmosis is considered as a cause of congenital ophthalmic anomalies.

4. Toxoplasmosis requires further investigation as a cause of primary adult ocular disease.

5. An increased number of substantiated diagnoses would point to the frequency and importance of the disease.

Henry, Pacific and Amity Streets.

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CORTISONE AND NEOSONE IN COMPLICATIONS FOLLOWING CATARACT SURGERY*

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In January and February, 1953, about 1,000 cataract extractions were completed at the Sir Henry Holland Eye Hospital, approximately one half of which were performed by the intracapsular method and one half by capsulotomy. The dressings of the capsulotomy patients were opened on the third day and those of the intracapsular patients on the fifth day. After the first dressing, the bandages were opened on alternate days, but those cases receiving cortisone or Neosone® were examined daily.

In this series of 339 cases an attempt was made to assess the value of cortisone and Neosone in treating some of the following common complications which occur after cataract surgery:

1. Iritis secondary to retained cortex and capsule.
2. Iritis secondary to blood in the anterior chamber.
3. Chemosis of the cornea possibly due to such agents as hydrarg perchloride, atropine, pontocaine, or neosynephrine or chemotherapeutic drugs used either before or directly after operation.
4. Photophobia and lacrimation of unknown origin without presence of cortex or capsule.
5. Striped or diffuse keratitis.
6. Iritis of unknown origin.

MATERIAL AND METHOD

The medicaments used were in the form of ophthalmic ointment supplied in tubes. The cortisone† contained 15 mg. of cortisone acetate per gm. The Neosone† contained the same amount, but had neomycin (5.0 mg. per gm.) incorporated in it. Ointment about two cm. in length was squeezed into the lower fornix of the eye, which was then closed and bandaged. The cortisone or Neosone, whichever was used, was applied daily until improvement was definite. Other drugs, such as atropine, in the form of drops were used simultaneously. Neither parenteral nor oral cortisone was given.

The number of applications of cortisone and Neosone and the complications for which they were used are outlined in Tables 1 and 2.

These tables show that, in a majority of patients, only a few applications of cortisone or Neosone were needed to produce a sig-

* From the Sir Henry Holland Eye Hospital.

® Neosone is the registered trademark for an ophthalmic ointment made by the Upjohn Company, Kalamazoo, Michigan.

† Cortisone and Neosone ophthalmic ointment were furnished by Upjohn & Company of Kalamazoo, Michigan.

TABLE 1

CORTISONE IN COMPLICATIONS OF CATARACT SURGERY

Complications and Incidence	Number of Applications					Total Cases
	1	2	3	4	5	
Iritis secondary to cortex and capsule	33	33	19	5	3	93
Blood in anterior chamber	31	26	8	5		70
Chemosis	3	7				10
Photophobia and lacrimation	24	10	3	1		38
Striped keratitis	14	7	6	1	7	35
Fresh blood in anterior chamber	6	4		2	1	13
Idiopathic iritis	5	1	2	2		10
Total No. Patients:						269

nificant and lasting effect. As may be seen in Table 1 under the action of cortisone in treating iritis secondary to cortex and capsule, 33 cases required only one application, 33 required two applications, and diminishing numbers required three or more applications. This is likewise true of cases of blood in the anterior chamber in which one or two applications caused a rapid resorption. The other complications show that one or two applications were sufficient to produce the desired therapeutic effect in most cases.

From the beginning of the investigation both doctors and nurses were favorably impressed by the rapidity of action of the cortisone and the quietness of the eyes after the first application, which was in contrast to the severity of some of the reactions and the prolonged photophobia and lacrimation previously experienced.

It had been thought that frequent applications were necessary in order to achieve the therapeutic effect. It was soon discovered, however, that this was not true. In our experience two or three applications of the amount already described was sufficient to exert and maintain the desired result. The outstanding clinical reaction was the rapid decrease in photophobia, lacrimation, and pain, even after only one application of cortisone or Neosone. It is not intended to imply that all complications can be cured with a few applications. Sufficiently marked improvement did occur after two or three applications to warrant discontinuing further use of cortisone and permit the natural physiologic processes of wound healing to take place. As soon as marked improvement occurred the cortisone or Neosone was discontinued.

It was a common occurrence to find a patient with photophobia, severe lacrimation, and conjunctival infection one day and, on the day following the first application of cortisone ointment, to find him able to open his eyes and look at his surroundings without difficulty. In fact, these patients opened their eyes as if their intracapsular extractions had

TABLE 2

NEOSONE IN COMPLICATIONS OF CATARACT SURGERY

Complications and Incidence	Number of Applications					Total Cases
	1	2	3	4	5	
Iritis secondary to cortex and capsule	2	10	4	1	1	18
Blood in anterior chamber	1	1	1	3	2	8
Chemosis	10	6	4	4		24
Photophobia and lacrimation	6	4	3			13
Striped keratitis		2	2			4
Fresh blood in anterior chamber		2				2
Idiopathic iritis	1					1
Total No. Patients:						70

been without complications. It was customary to find an eye, which, previously, had been red and angry, white and tranquil without any signs of spasm or lacrimation.

For those patients who showed evidence of mild conjunctivitis or chemosis as well as iritis, Neosone was found to be most efficacious. It should be pointed out that all cases in this clinic are operated without previous bacteriologic studies. Due to economic pressure, the majority of poor patients who come to the hospital will stay no more than 10 or 12 days; therefore, all clinically clean eyes are submitted to operation. The incidence of intraocular infection was 0.2 percent; however, a number of cases of conjunctivitis were observed following operation. Sometimes Neosone was combined with silver nitrate (two-percent drops, daily).

No definite effect of cortisone was observed during studies of a series of cases of striped keratitis.

Some instances of delayed wound healing did occur in this series, but there were other cases in which wound healing seemed to be accelerated after the application of cortisone. Since, however, delayed wound healing occurred with approximately the same frequency in those patients who did not receive either cortisone or Neosone, we feel that surgeons who wish to treat the after complications of cataract extraction with cortisone need have no fears of an adverse effect on wound healing. In our series delay in wound healing seemed mostly to occur in those patients of poor economic circumstances who were suffering from nutritional and vitamin deficiencies.

The hospital stay of the patients who had the complications was not lengthened, as is usually observed; indeed, it was shortened since blood and cortex absorbed more rapidly.

Summary and Conclusions

1. A total of 339 patients, who presented some of the common complications following cataract extractions, were treated with cortisone ophthalmic ointment.

2. The patients were observed once a day, then cortisone ointment was applied and the progress was noted.

3. Cortisone and Neosone were found to have a beneficial effect in abolishing the iritis and symptoms associated with the retention of cortex and blood in the anterior chamber (complications of capsulotomy extractions).

4. Postoperative discomfort and hospital stay were shortened by use of cortisone and Neosone.

5. Wound healing did not appear to be affected by the treatment.

6. The drug had no apparent influence on striped keratitis.

7. The therapeutic effect can be produced and maintained by application at 24-hour intervals.

Sir Henry Holland Eye Hospital.
238 Quincy Street.

We gratefully acknowledge the assistance of Mr. B. Fazl Din in making this study.

Ophthalmic Miniature

Sidmouth, May 15, 1852.

Lord Buckinghamshire will thank Mr. Dixey to send him down to Sidmouth a pair of spectacles such as he thinks will suit a youth of 16 years of age, who has never worn any, but is so shortsighted as to be obliged to hold his face close to his plate when he takes his meals. They are wanted for one of Lord B's sons.

From the correspondence files of Dixey's, London, England.

VITREOUS HEMORRHAGE AND RETINOPATHY ASSOCIATED WITH SICKLE-CELL DISEASE*

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Spontanenous vitreous hemorrhage, first thoroughly described by Henry Eales[7] in 1880, is now defined as a recurrent intraocular hemorrhage of unknown cause usually occurring in young men of apparently good health.[6] Causes have been ascribed to systemic infections, toxins, trauma, and focal infections in the teeth, sinuses, or tonsils. Other causes described are syphilis, diabetes, and cardiovascular disease; calcium deficiency with diminished parathyroid secretions;[23] endocrine imbalance (adrenals); blood dyscrasias; circulatory or trophic disturbances (Buerger's disease); and tuberculous periphlebitis of the retinal vessels.[19]

Verhoeff[23] has stated that the hemorrhages result from engorgement of the capillaries and the small veins. When this engorgement is severe and persistent, new vessels form which may extend into the vitreous. At the same time hemorrhage into the vitreous occurs. The extravasated blood causes a stimulus to endothelial-cell proliferation which becomes organized by new vessels, producing the typical retinitis proliferans. Usually the lesions are large, round, or irregular retinal hemorrhages, which are confined to the periphery of the retina near the veins. The visual loss is proportionate to the vitreous opacification which frequently clears rapidly between attacks.

In sickle-cell disease, hemorrhagic lesions and infarcts attributable to the sickling process have been described as occurring in the reticulo-endothelial system, kidneys, lungs, joints, cerebrum, subarachnoid, intestines, and so forth.[1, 4, 5, 10, 14, 16, 17, 22, 24]

The sickling phenomena have been shown to be dependent solely upon the erythrocyte and not the plasma.[10, 21] When placed in a low oxygen atmosphere, the sickle-cell hemoglobin of the erythrocyte is reduced. In this less soluble state, long thin rodlike particles of the reduced sickle-cell hemoglobin assume a parallel and equidistant arrangement.[13]

These crystals give the characteristic sheaf or sickle shape to the intact erythrocyte which is now two to five times longer than the original diameter with tapering processes from each end. This sickling process is reversible for, upon reoxygenation, the cells will assume a normal contour.[11]

Unlike erythrocytes of normal contour, sickled cells are rigid and inflexible. In smaller capillaries where normal cells repeatedly alter their shape for passage, the rigid sickled cells become impacted and stasis with thrombosis and hemorrhage results.[17, 21] This causes a further decrease of the oxygen supply to the local vasculature with ensuing endarteritis and infarction of the area.[15, 24]

Once this process has started, the thrombosis and hemorrhage secondary to sickling in the various body organs is identical with Verhoeff's theory for retinal hemorrhages and the resulting retinitis proliferans. With such hemorrhagic lesions occurring in almost every other organ of the body, it should be expected that the retinal vessels also may be involved.

Edington and Sarkies, in 1952, reported from Africa two cases of sickle-cell anemia with retinal microaneurysms, and a vitreous hemorrhage in one.[8] In discussing a case

* From the Departments of Ophthalmology and Hematology, Veterans Administration Hospital. A preliminary report was presented before the Chicago Ophthalmological Society, January 21, 1952.

of subarachnoid hemorrhage in a patient with sickle-cell anemia in 1930, Cook mentioned retinal hemorrhage among the physical findings;[4] and Henderson, in 1952, tabulated retinal hemorrhage as a presenting sign or symptom in one of the 54 cases of sickle-cell anemia studied.[14]

The only other reference to an ocular pathologic process found in the literature was that first reported by Harden, in 1937, describing dilatation of the retinal vessels with marked venous tortuosity in two Negro children with sickle-cell anemia.[12]

Our attention was first drawn to the presence of intraocular hemorrhage in a patient with the sickling phenomenon in September, 1951. A study of an additional 28 patients was made to determine if the occurrence of such retinal vascular pathologic processes and the sickling process were or were not related.

None of these patients demonstrated any foci of infection relative to the ear, nose, and throat, dental, genito-urinary, or pulmonary systems. Systemic diseases other than sickling were not evident, and metabolic studies revealed no disturbances of calcium or carbohydrate metabolism. No history was obtainable of past trauma or exposure to toxins which could account for the retinal changes.

Relative to the sickling phenomena, all patients received the following laboratory studies: direct blood smear for sickling; sodium metabisulfite preparations for sickling;* red, white, differential, and reticulocyte counts; hemoglobin determination; sedimentation rates; serum Van den Bergh, both direct and indirect; urine urobilinogen excretions; and red-cell osmotic fragility.

In addition, the majority also had fetal hemoglobin determinations made by the method of Singer,[20] liver profiles, roentgen bony surveys, urologic and renal function studies. In a few of these patients, electrophoretic hemoglobin studies were also made.†

Those individuals who met three or more of the following criteria were classified as having sickle-cell anemia and those failing were classified as having sickle-cell trait: (1) Red blood count, less than 3.5 million; (2) hemoglobin, less than 11.0 gm.; (3) total serum bilirubin, greater than 1.0 mg. and/or a two-hour Watson, greater than 0.25 or 1.0 Ehrlich unit; (4) fetal hemoglobin, greater than 1.7 percent; (5) reticulocyte count, greater than 1.0 percent.

With very few exceptions those classed as sickle-cell anemia met all of these criteria, and those classed as sickle-cell trait failed to meet any of them. Accordingly in the group of 29 patients, 19 were classed as sickle-cell trait, and 10 as sickle-cell anemia. In nine of these patients, ocular pathologic findings are reported in the case studies which follow: Cases 1, 2, 7, 8, and 9 were diagnosed as sickle-cell disease with anemia and Cases 3 through 6 were diagnosed as sickle-cell disease without anemia (sickle-cell trait).

Case reports

Case 1

R. W., a Negro, aged 29 years, had vision of: R.E., 20/300; L.E., 20/300. With correction, it was improved to R.E., 20/20; L.E., 20/60.

The disc and macula of the right eye were normal. There were five areas of varying size, the largest being 1.5 disc diameters, of thin, veil-like, elevated, white fibrous tissue covered by a fine network of capillaries.

The vitreous of the left eye contained red blood cells and, although poorly visualized, there were round hemorrhages in the temporal periphery of the retina.

Within three months the vitreous cleared sufficiently to visualize several absorbing

* This material was kindly supplied by Eli Lilly and Company, Indianapolis, Indiana.

† These studies were kindly done by Dr. Karl A. Singer and his co-workers in the Hematology Research Division of the Michael Reese Hospital, Chicago.

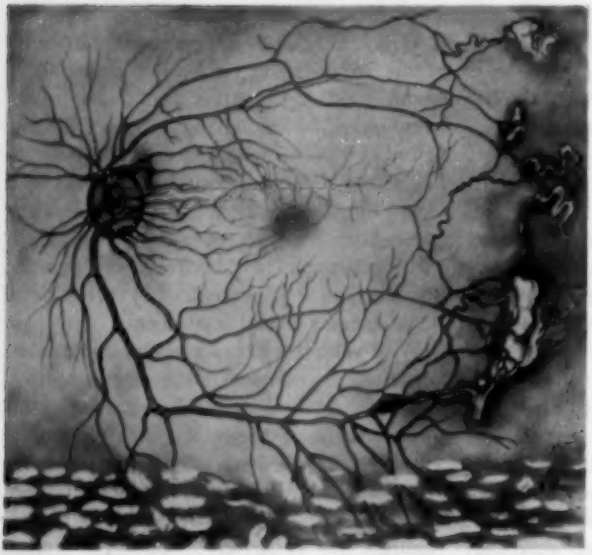

Fig. 1 (Henry and Chapman). Case 1. Fundus of the left eye, illustrating retinal hemorrhage, retinitis proliferans, and absorbing vitreous hemorrhage (ghost cells).

areas of subhyaloid and retinal hemorrhages, as well as retinitis proliferans. In the dependent portion of the vitreous, degenerated red blood cells (ghost cells) had taken on the more solid white appearance of fibrous tissue without neovascularization.

Case 2

C. G., a Negro, aged 28 years, had vision of: R.E., 20/300; L.E., light perception. With correction, it was improved to: R.E., 20/40; L.E., light perception.

The disc and macula of the right eye were normal. An area of retinitis proliferans was seen in the temporal periphery and multiple areas of chorioretinal atrophy in the peripheral lower half of the retina. The vitreous of the left eye contained red blood cells and, in the temporal periphery, was an area of chorioretinal atrophy.

Case 3

D. T., a Negro, aged 29 years, had vision of: R.E., 20/20; L.E., absent.

The vitreous of the right eye contained grayish, irregularly dispersed debris in its dependent portions which resembled ghost cells. The macula and disc were normal. In the temporal periphery of the retina was a large pigmented area of chorioretinitic atrophy.

The surgical specimen of the left eye, removed elsewhere, was reported by the Armed Forces Institute of Pathology as showing sicklemia, recurrent retinal hemorrhage, absorbing vitreous hemorrhage, and retinal detachment.

Case 4

W. D., a Negro, aged 45 years, had vision of: R.E., 20/15; L.E., 20/100, with correction, 20/25.

The right fundus was normal. The disc and macula of the left eye were normal. Along the course of the inferior temporal artery was a hemorrhage, one by three disc diameters in size, subhyaloid in position.

Case 5

D. P., a Negro, aged 39 years, had vision of: R.E., 20/20; L.E., 20/15.

The right fundus was normal. The disc and macula of the left eye were normal. Several fluffy white patches and small round

subhyaloid hemorrhages were seen in the temporal retina.

CASE 6

J. S., a Negro, aged 31 years, had vision of: R.E., 20/15; L.E., 20/15.

The macula of the right eye was normal. Adjacent to the disc was a filmy white mass which followed the course of the inferior temporal vein. The left fundus was normal.

CASE 7

R. E., a Negro, aged 29 years, had vision of: R.E., 20/20; L.E., 20/20.

The right fundus was normal. The disc and macula of the left eye were normal. In the nasal retinal periphery was a pigmented area of chorioretinal atrophy, one disc diameter in size.

CASE 8

F. C., a Negro, aged 27 years, had vision of: R.E., 20/40; L.E., 20/40. With correction it was improved to: R.E., 20/20; L.E., 20/25.

The disc and macula of the right eye were normal. Encircling the entire periphery of the retina was chorioretinal atrophy with pigment migration, being most concentrated in the superior temporal area. The disc and macula of the left eye were normal. Along the course of the superior temporal artery was a single pigmented atrophic lesion.

CASE 9

L. B., a Negro, aged 34 years, had vision of: R.E., 20/20; L.E., 20/20.

The disc and macula of the right eye were normal. The retinal vascular tree was dilated and tortuous. The disc and macula of the left eye were normal. In the temporal periphery were three areas of glial proliferation.

DISCUSSION

Cases 1 through 5 had retinal hemorrhages; Cases 1, 2, and 3 had vitreous hemorrhages; and Cases 1, 2, 5, and 6 had

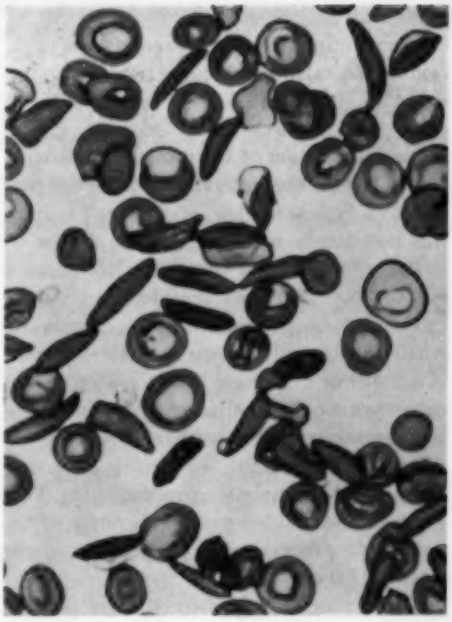

Fig. 2 (Henry and Chapman). Sickle cells, as demonstrated in a patient with sickle-cell disease without anemia by the rapid sodium-metabisulfite method.

retinitis proliferans. The difference in these lesions is one of degree usually due to capillary or venous occlusion.

It has been shown pathologically that varices and aneurysms develop following venous occlusion, and hemorrhage into the vitreous frequently occurs.[9] Focal necrosis may occur in scattered areas causing chorioretinal atrophy as seen in Cases 2, 3, 7, and 8.

Retinal detachment, as in the pathologic specimen of Case 3, is due to the accumulation of fluid subretinally. This fluid is the result of the marked edema of the affected tissues and may also accumulate within the retina forming large irregular cystic spaces.

Case 9 illustrates the results of arterial occlusion causing retinal infarcts. The affected portion of the retina undergoes cloudy swelling which progresses to autolysis, phagocytosis, and finally heals with prolif-

eration of glial tissue.

Chorioretinal atrophy as seen in Cases 2, 3, 7, and 8 may also be due to infarcts within the choroid. This type of lesion can produce a subretinal hemorrhage as seen in Coats' disease. Dilatation of the retinal vessels with venous tortuosity referred to in the literature as a typical finding in sickle-cell anemia was noted only in the right eye of Case 9.

Bauer,[2] on several occasions, has pointed out the occurrence of symptoms of sickle-cell crises in patients without anemia or what was formerly known as sickle-cell trait. A number of cases have been reported within recent years of hematuria occurring in sickle-cell disease with a considerable number of these patients having no evidence of any anemia or hemolytic process.[1, 5, 10] This gives further evidence of the severity of the pathologic process which may occur in the so-called "trait."

In the nine cases presented, four patients had no evidence of an anemia or hemolytic process; nevertheless, the severity of their ocular lesions did not differ from those with anemia. We agree with Bauer[2] in his statement: "That the disease known as sickle-cell anemia might better be named sickle-cell disease, because anemia, though the best known . . . sign of this disease, is not the essential and not the most dangerous one."

Summary

A cause of Eales' disease is presented which will account for a number of cases of unknown etiology. Four cases of retinal lesions were found in a review of the literature, only two of which were ascribed by the authors to sickle-cell disease. We submit nine cases with sickle disease, both with and without anemia, and associated retinal pathology. It is further suggested that other idiopathic vascular pathologic processes occurring in Negroes, such as Coats' disease, may be due to sickle-cell disease.

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THE SIGNIFICANCE OF THE UNILATERAL ARGYLL ROBERTSON PUPIL*

Part II. A critical review of the theories of its pathogenesis

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Introduction

In a recent investigation[105] of neurosyphilitic patients with the slitlamp biomicroscope, an unusually high incidence of unilateral Argyll Robertson pupils was found. The investigator also noted the concomitant abnormalities of the iris which other observers[21, 25-26, 35, 49, 62, 68-69, 89] have found in association with the Argyll Robertson syndrome. These two characteristics of the Argyll Robertson syndrome are inconsistent with a central nervous-system pathogenesis of the syndrome since neurophysiologists and pathologists concur that a unilateral miotic, light-rigid pupil associated with the reported abnormalities of the iris could not be explained by any known central nervous-system lesion.

Despite this discrepancy between theory and fact, authoritative opinion of the past three decades has favored a central nervous-system pathogenesis for the Argyll Robertson pupil.[7, 28, 41, 44-45, 63, 71, 83-84, 92, 100, 103-104] This opinion has been influential in directing experimental research and pathologic studies toward finding a lesion in the central nervous-system that would explain the miotic light-rigid pupils which are found in neurosyphilis.

This search, though intensive, has failed to demonstrate any lesion that could explain or produce the complete Argyll Robertson syndrome. Therefore, these authorities have postulated the existence of two separate lesions; one to explain the miosis and one to explain the light-rigidity.

* From the Department of Ophthalmology, Northwestern University School of Medicine. Part I of this paper appears in The Journal, **38**: 34 (July) 1954.

Despite this concession, no central nervous-system lesion that could explain even the light-rigidity alone has been found consistently in neurosyphilitic patients with the Argyll Robertson syndrome, although such a lesion has often been demonstrated in diseases other than syphilis. It is notable that, in these other diseases, the light-rigidity occurs alone without miosis. The light-rigidity is accompanied by miosis only in neurosyphilis and for these cases no central nervous-system lesion has been found.

On the other hand, clinical and laboratory studies support the theory that a lesion intrinsic in the iris is responsible for all the characteristics of the Argyll Robertson syndrome. Moreover, the literature concerning the actions of the iris musculature makes it apparent that a lesion in the iris itself could explain the simultaneous occurrence of miosis, immobility to light and to pain and to darkness, as well as overactivity to accommodation-convergence.[2, 11, 16, 18-19, 22, 25-26, 30, 37-39, 42-43, 51, 55, 60-61, 72, 77, 82, 95, 99]

It should be noted that all these signs appear simultaneously only in neurosyphilis, although each sign may occur separately in other diseases. This complete Argyll Robertson syndrome, therefore, is worthy of consideration in its entirety in any theory of its pathogenesis.

In view of the failure of the investigations of the central nervous system to demonstrate a lesion that could explain the syndrome, it seemed worthwhile to review the history of the theory of a central nervous-system pathogenesis of the Argyll Robertson syndrome. Furthermore, on the basis of the evidence that supports a lesion intrinsic in the iris of patients with an Argyll Robertson syndrome, it seemed advantageous to attempt to explain how this lesion of the iris could result in a complete Argyll Robertson syndrome, whether bilateral or unilateral.

Review

In 1869, Douglas Argyll Robertson[3, 4] described a syndrome of pupillary abnormalities which he called spinal miosis. He specified five conditions which were present in all of the four cases he studied. The pupils of both eyes were less than two mm. in diameter, they constricted actively to accommodation-convergence but not to light no matter how strong the stimulus; they did not dilate fully when atropine solution was instilled into the conjunctival sac; they did not dilate when the patient was in pain. In all, the retinas were sensitive to light.

It was not until 1897 that the discovery was made that the disease these patients had was syphilis of the nervous system. Since then, the search for the lesion responsible for this syndrome has fired the imagination of countless clinicians, physiologists, and neuropathologists. (References 1 to 104 constitute a partial list.)

Despite the widespread interest in the problem, however, careful studies of the midbrain have not yet proved the presence of a lesion in a location that could cause both the miosis and the light-rigidity of the Argyll Robertson pupil. Moreover, there is no possible central nervous-system lesion that could explain the presumably rare unilateral Argyll Robertson syndrome.[7, 9, 12-13, 15, 24, 30, 37-38, 45, 55, 63, 67, 70-71, 76, 83, 85, 88, 92-96, 100, 102-104]

These facts led Kinnier Wilson[102] (1921) to propose that the retention of the pupil reaction to accommodation associated with a loss of the reaction to light was, alone, a sufficiently notable characteristic of tabetic pupils to allow disregarding the miosis. This prominent neurologist advised, therefore, that pathologists search for a lesion that would explain this single Argyll Robertson sign without regard for the miosis or other characteristics of the Argyll Robertson syndrome.[103, 104] He believed that the miosis could be explained only on the basis of a separate central nervous-system lesion.

In 1926, Behr[7] agreed with Wilson that the miosis could be explained only by a separate lesion but he believed that the miosis was a necessary criterion in a clinical diagnosis of Argyll Robertson pupils. Despite this ad-

vice by Behr, clinicians, influenced by Wilson's dictum, reported their findings in patients with the single Argyll Robertson sign in one or both eyes associated with large pupils.[1, 5, 10, 12, 15, 20, 23, 27-28, 31, 34, 36, 40-41, 48, 58, 56-59, 64, 69, 73-75, 78, 80-81, 86-87, 90, 94, 97-98, 101]

Many of these patients were treated for syphilis despite negative serologic or spinal-fluid tests.[5, 15, 34] Most of them eventually developed gross signs of other diseases such as multiple sclerosis, diabetes, or tumor in the region of the superior colliculus. When the brains of any of these patients came to the pathologist, the cause of the Argyll Robertson sign with dilated pupils was readily discerned (fig. 1).

The afferent fibers of the pupillary light reflex were atrophied by pressure or were inactive from a demyelinating plaque in the region between the brachium of the superior colliculus and the Edinger-Westphal nucleus.

In consequence of the decussations in the posterior commissure, both eyes were equally affected by a unilateral lesion and direct and consensual reactions to light were absent. The accommodation-convergence reaction was not affected, however, and constriction of the pupil did take place. These pupils were large, since light is the ordinary stimulus that keeps the pupil from assuming the dilated state.

In textbooks,[24, 104] numerous diseases were listed in which the pupils show the dichotomy between the light and accommodation reactions and which were said to show Argyll Robertson pupils without syphilis. Actually, these pupils never show the whole syndrome but merely the light-rigidity or Argyll Robertson *sign,* since in not a single case is miosis also present. On the other hand, when miosis accompanies the light-rigidity, this combination is pathognomonic of syphilis.

Adie,[2] Behr,[7] Duke-Elder[24] agree that this complete syndrome is found in no disease other than syphilis. They are divided, however, on the question of whether the whole syndrome is necessary for a diagnosis of tabetic pupils. This division of opinion among the authorities is unfortunate.

Although it is true that there is no longer danger that a patient with mydriasis and light-rigidity will be treated for syphilis in the absence of a positive serologic test for syphilis, a more subtle danger remains, when the textbook writers continue to state that light-rigidity alone is characteristic of neurosyphilitic pupils. A syphilitic patient with this sign alone might well have some cause for his pupillary abnormalities other than his syphilis.

Consideration of the manner in which the Argyll Robertson syndrome develops will demonstrate that large, light-rigid pupils are never a part of it and that the miosis cannot be disregarded:

First, under the slitlamp biomicroscope, according to Lowenstein,[60, 61] there is an increase in the fatigability of the light reflex. No other changes were apparent grossly at this time but other signs appeared after a year of observation. Then, the diameter of the dark-adapted pupils decreased and the speed of the light response was reduced. Irregularities in the margin of the pupil and a segmental atrophy of the iris appear at this time according to McGrath[68] (fig. 2). The light reflex disappears in the atrophic sectors of the iris although it remains in the more normal sectors.

Careful students of this problem, McGrath,[68] Myerson and Thau,[77] Langworthy and Ortega,[55] Adie,[2] agree that the light response does not completely disappear until the diameter of the pupil has decreased to less than 2.5 mm. As long as the pupil is larger than this, it can be seen to dilate in the dark and to constrict in the light although these responses become more sluggish as the diameter of the pupil decreases.[8]

Both eyes are always affected, but rarely equally so. Until the pupil has become miotic, the light and accommodation reactions are retained and the pupil is not the Argyll Robertson one. This sequence of events is, indeed, pathognomonic of neurosyphilis.

A syphilitic patient who has a complete

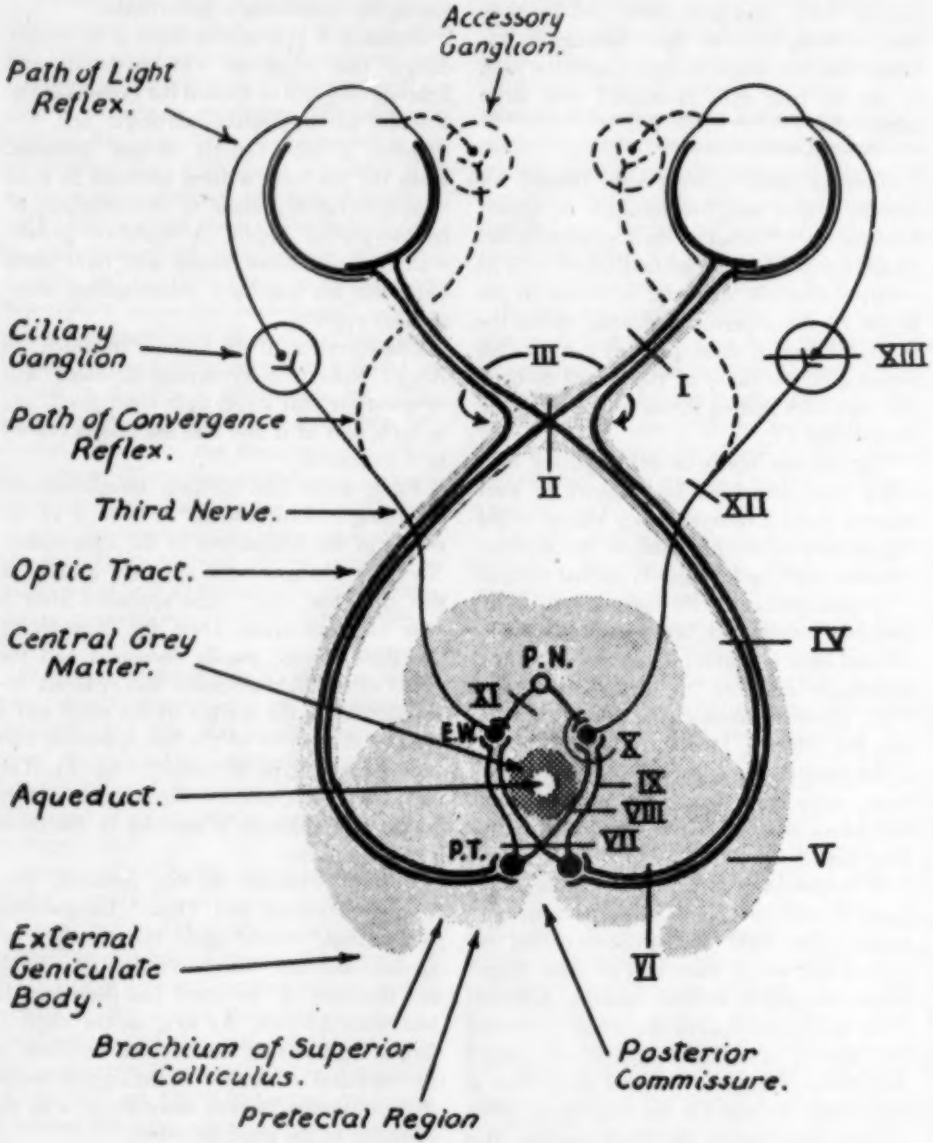

Fig. 1 (Apter). THE PUPILLARY PATHWAYS FOR THE LIGHT REFLEX

P.N., Perlia's nucleus. E.W., Edinger-Westphal nucleus. P.T., Pretectal nucleus.

The roman numerals denote lesions accompanied by the following symptoms:

I. Optic nerve: *unilateral amaurotic paralysis* (abolition of the direct reaction on the ipsilateral side and the consensual on the contralateral side: retention of the consensual on the ipsilateral side and the direct on the contralateral side). Retention of the near reflex and the lid reflex.

II. Medial chiasma: *bitemporal hemianopic paralysis.*

(Continued on facing page)

Argyll Robertson *sign* alone, without miosis, should be studied thoroughly for some cause of his pupillary abnormalities other than his syphilis.

To be sure, a syphilitic lesion such as a gumma or meningo-encephalitis may be responsible for other pupil disturbances; but such other disturbances are not related to the syndrome, although they may include the Argyll Robertson *sign*. However, when the Argyll Robertson *sign* occurs alone without miosis even it is not related to the complete syndrome.

The pathologic process causing the Argyll Robertson *sign* is readily discoverable. It may occur as shown in Figure 1, at levels VII, VIII, XIII. In sharp contradistinction, however, careful studies of brains of patients in whom the complete Argyll Robertson syndrome was found have failed to reveal a consistent pathologic condition of the central nervous-system. Furthermore, experimental neurophysiologists have rejected all hypotheses which endeavor to localize such a lesion.

The miosis cannot be explained by damage to the components of the sympathetic nervous system of the midbrain, as Merritt and Moore do,[71] since such miosis must always be bilateral. Nor can irritation of the parasympathetic nerve fibers explain the miosis because atropinization of the iris, with its consequent release of the iris muscles from the action of the parasympathetic nerves, does not result in the slow, imperfect dilation characteristic of Argyll Robertson pupils. Moreover, Ranson and Magoun have shown that an irritated pupilloconstrictor mechanism would not result in the permanent light-rigid miotic pupils of neurosyphilis.[83]

Continued failure to demonstrate injury to these pathways in syphilis or to produce the Argyll Robertson syndrome by experimental lesions make these hypotheses untenable.

Unilaterality is another characteristic of the Argyll Robertson syndrome that has been overlooked. Since statistics[13-14, 17, 21, 32, 45, 47-48, 63, 70-71, 91] have heretofore disclosed that the unilateral miotic light-rigid pupil of neurosyphilis occurs rarely, this oversight has seemed justifiable to many theorists.

A review of the statistics on which the incidence of the unilateral syndrome is based makes it apparent that this oversight should be scrutinized.

In 1936, Merritt and Moore[71] reported on their survey of charts of 749 cases of neurosyphilis taken from the files of the Boston City Hospital. They found that unilaterality was mentioned in eight of them, or 1.4 percent and bilaterality in 30 percent. In very few of the 749 charts were the criteria for pupil diagnosis mentioned. Merritt and

←

III. Lateral chiasma: *binasal hemianopic paralysis.*

IV. Optic tract: *contralateral hemianopic paralysis* (Wernicke's reaction).

V. Lesion of the proximal part of optic tract: normal pupillary reactions.

VI. Superficially in the region of the brachium and tectum: *contralateral hemianopic paralysis.*

VII. Central decussation: *bilateral reflex paralysis*—inactivity to light (direct and consensual) with retention of the near reflex, the lid reflexes and the psychosensory reactions (*bilateral Argyll Robertson pupil*) (according to Behr).

VIII. Between the decussation and the constrictor center: ipsilateral abolition of direct and consensual reactions with retention of both contralaterally—*unilateral Argyll Robertson pupil* (according to Behr).

IX. A partial lesion corresponding to VIII: ipsilateral abolition of the direct reaction with retention of the consensual reaction; retention of both contralaterally.

X. Nuclear or extensive supranuclear lesion: ipsilateral absolute pupillary paralysis.

XI. Isolated abolition of near reflex (the *inverse Argyll Robertson pupil*).

XII. Lesion of IIIrd nerve: absolute pupillary paralysis.

XIII. Lesion of ciliary ganglion: abolition of the light reflex with retention of the near reflex (*Argyll Robertson pupil*).

(Reprinted with permission of the publishers and Sir Stewart Duke-Elder from *Textbook of Ophthalmology*, London, Henry Kimpton, 1949, v. 4, Figure 3139.)

Moore published these results on the incidence of the unilateral Argyll Robertson pupil with the warning that the limitations of their study be recognized. Unfortunately, this caution was not heeded by most reviewers so that 1.4 percent continues to be quoted as the incidence of the unilateral Argyll Robertson pupil in neurosyphilis.

Duke-Elder, Walsh, Behr, and others, nevertheless, have attempted to include unilaterality in their consideration of the pathogenesis of the Argyll Robertson pupil. Since these observers have chosen to disregard the miosis of the Argyll Robertson syndrome, however, this inclusion of unilaterality does not have meaning enough to help solve the problem of the pathogenesis of the complete syndrome.

They place the lesion responsible for the unilateral Argyll Robertson pupil as seen in Figure 1, level VIII. There is no disagreement that interruption of pathways here would cause a unilateral Argyll Robertson *sign*. However, a lesion here cannot explain the sign if it occurs simultaneously with miosis.

The fact is that there is no central nervous-system lesion that could possibly produce a unilateral Argyll Robertson syndrome—a pupil characterized by miosis and light-rigidity as well as by an overactive accommodation constriction. Therefore those (Merritt and Moore) who include miosis in the requisites of an Argyll Robertson syndrome and who insist on a central nervous-system pathogenesis are forced to disregard the unilaterality; and those who include unilaterality in their considerations must disregard the miosis in order to place the pathologic process in the central nervous system.

The miosis, light-rigidity, and unilaterality do occur together in neurosyphilis, however. Moreover, coexistence of these three signs appears often if these neurosyphilitic patients are examined under the slitlamp biomicroscope.

In a recent study[105] I described one case of tabes in which the slitlamp biomicroscope technique for examining these pupils was discovered accidentally. Using this technique on 46 neurosyphilitic patients, I found 13 patients with the unilateral Argyll Robertson syndrome. In these 46 patients there was not one instance of the bilateral syndrome. In all the cases, both eyes were involved and the pathologic process was developing into an Argyll Robertson syndrome. In all, however, the process had become fully developed in one eye before it had in the other so that one eye was miotic and light-rigid while the other was not.

These findings lead to the conclusion that the same lesion may result in either a unilateral or a bilateral case of the syndrome. The lesion produces the unilateral syndrome during a phase in which one pupil has reached the end-stage and one pupil is approaching it. This same lesion produces the bilateral syndrome after both pupils have progressed to the end-stage. The fact that syphilitic patients are discovered early and treatment arrests the pathologic process in this earlier stage may be partly responsible for the high incidence of unilateral cases reported in this recent study.[105]

The biomicroscopic findings support the contention of McGrath,[68] Langworthy,[55] Myerson,[77] Biyadoux,[8] and Adie[3] who have separately maintained that the Argyll Robertson pupil does not lose its light reaction until the miosis is marked. Since the miosis and the light-rigidity progress at the same rate, it is logical to assume that a single lesion is responsible for both signs.

There is no need for the dichotomous viewpoint held by many authorities if the pathologic process producing this syndrome is sought for in the peripheral nervous system rather than in the central nervous system. A review of the findings of those who have proposed a peripheral nerve pathogenesis of the Argyll Robertson syndrome, therefore, seemed worth while.

The prevalence of gross iris abnormalities in patients who show the Argyll Robertson syndrome has been the main support of those

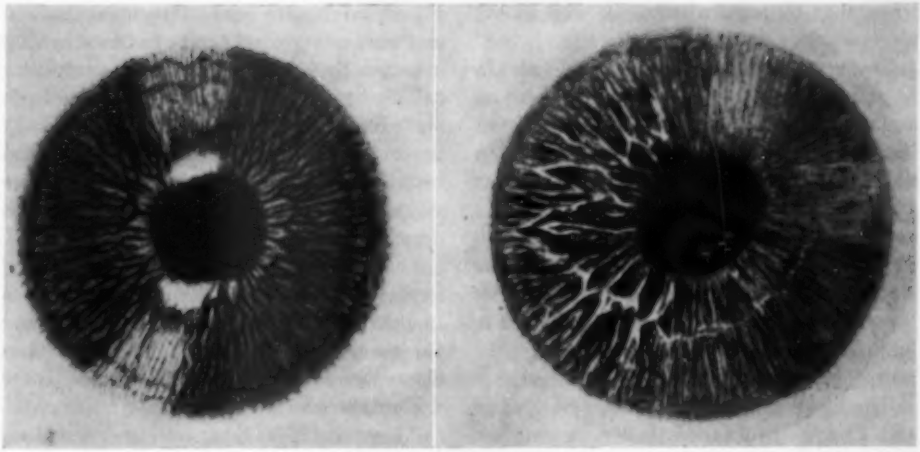

Fig. 2A (Apter). Drawings of partially abnormal irises in which the abnormal sectors did not respond to light incident on the retina, whereas the more normal sectors did respond.

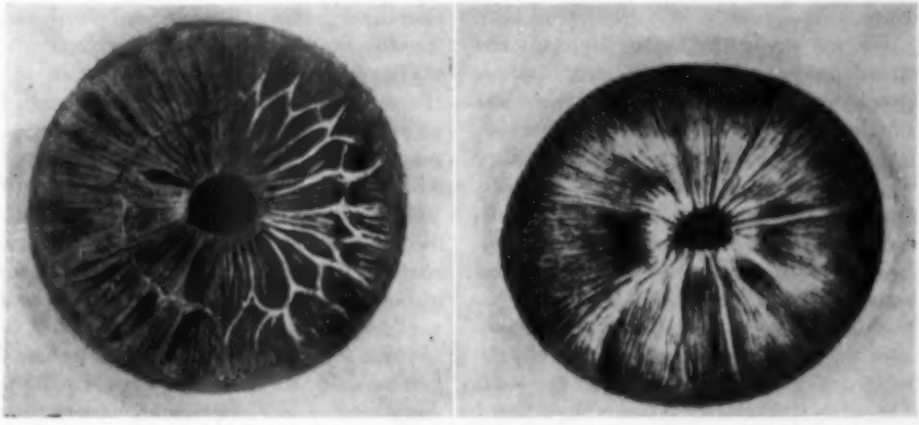

Fig. 2B (Apter). Drawings of abnormal irises in patients with Argyll Robertson syndrome. (Reproduced with permission of the publishers from McGrath.[46])

who have sought a peripheral nerve lesion to explain the syndrome. An interruption of sensory or motor pathways central to the ciliary ganglion could not result in these iris abnormalities. Only a peripheral nerve lesion could produce them.

Marina,[67] therefore, has searched for a lesion in the ciliary ganglion in patients with neurosyphilis. He attempted to demonstrate spotty degeneration in this ganglion in patients who were developing an Argyll Robertson pupil. His description of this degeneration is not convincing, however. Moreover, he postulated an earlier pathologic condition in the sympathetic nervous system to explain the miosis. The attempts of others (Mantoux,[65] Dupuy-Dutemps[25]) to substantiate Marina's findings of abnormalities in the ciliary ganglion have failed.

On the other hand, Dupuy-Dutemps,[26] and Lowry[62] have demonstrated by gross and by histologic study of these abnormal

irises that the musculature, as well as the blood vessels and stroma, is altered.

The gross abnormalities consist in an absence of the collarette and a replacement of the normal crypts by fine radial striations. Near the pupillary margin the iris may be thin enough to allow transillumination. These abnormalities develop slowly, appearing in one or more sectors of the iris at first and then involve more and more sectors until the whole iris is altered. McGrath noted that these abnormal sectors failed to respond to light incident on the retina even though the normal sectors retained the light response.

The histologic studies by Dupuy-Dutemps and Lowry have failed to demonstrate the precise mechanism by which the abnormalities of the iris produce a pupil inactive to light and pain but hyperactive to accommodation.

The iris studied by Dupuy-Dutemps had no inflammatory changes. There was simple atrophy of the stroma connective tissue. The muscle fibers of the sphincter pupillae were fewer and finer than normal. Large clumps of pigment were present among the radially arranged spindle-shaped cells that are currently named the dilator muscle.

On the other hand, Lowry's study of sections of these irises showed an increase in plasma cells throughout the iris. He postulated that these were found in the fully developed Argyll Robertson syndrome only. The early pathologic, process he believed, is a cuffing of blood vessels in the iris analogous to the general pathologic degeneration of neurosyphilis.

It must be recognized that neither of these studies gives conclusive evidence concerning the pathogenesis of the abnormal iris nor concerning how the abnormal iris results in the signs of the Argyll Robertson syndrome.

On the basis of the grossly abnormal irises and of the lack of support for a central nervous-system pathogenesis, Langworthy and Ortega[88] postulate that a lesion in the nerves intrinsic to the iris is responsible for the iris atrophy and abnormal reflexes of an Argyll Robertson pupil. They base their explanation on their findings in the iris of normal rabbits in which they found three types of nerves: (1) Sympathetic fibers to the blood vessels, pigment cells, and stroma of the iris; (2) proprioceptive sensory fibers to the blood vessels near the pupillary margin; (3) parasympathetic motor fibers to the sphincter muscle.

Langworthy and Ortega advance several hypotheses concerning the manner in which lesions in each nerve supply might account for the Argyll Robertson syndrome. They discard each separate lesion as inadequate to explain the whole syndrome.

They conclude that a lesion to blood vessels is probably primary in the iris, as it is in the spinal cord in patients with tabes. Following partial occlusion of diseased blood vessels, the nerves become atrophied and then the weakened musculature results.

They believe that the sphincter pupillae is at rest in miosis. The fact that the normal pupil is miotic in sleep supports their assumption. On the other hand the early postmortem dilation of both normal and Argyll Robertson pupils appears to refute this theory. Langworthy and Ortega, however, attribute the dilation after death to emptying of the blood vessels. This does seem to be a reasonable hypothesis.

These authors do not explain adequately the retained accommodation reflex of the Argyll Robertson syndrome, however. They believe that the accommodation reflex must be more powerful than the light reflex in order to overcome the inertia of the weak sphincter pupillae. This would not explain the overactive constriction that takes place when patients with an Argyll Robertson syndrome accommodate for near vision.

A review of the literature has uncovered the fact that no theorist has explained this hyperactive accommodation response of the Argyll Robertson pupil. I have found, however, that the Helmholtz treatise on normal accommodation[89] could help explain this sign of the Argyll Robertson syndrome.

The Helmholtz theory states that in the normal eye the root of the iris is moved forward during constriction of the ciliary muscle. This causes the whole iris to be displaced toward the center of the pupil thereby resulting in constriction of the pupil. Donders[22] concurred with this theory and Coccius[18, 19] and Landolt[51] demonstrated that the whole iris moves centripetally in accommodation.

This movement toward the center of the pupil is most effective when the pupil is small before the accommodation response of the ciliary muscle takes place. This forward movement of the root of the iris results in a constriction of the pupil. It takes place independent of the nerves of the iris.

When this theory of normal accommodation is applied to the miotic pupil and atrophic iris of the Argyll Robertson syndrome, it seems logical that the centripetal movement of the whole iris, which accompanies the forward movement of the root of the iris, might produce a greater effect than in the normal eye. Since this action is independent of the innervation to the iris, the response could take place despite diseased iris nerves. Thus, the pupil that is immobile following all other stimuli is excessively active upon accommodation.

I wish to emphasize that no studies have been made on the iris of the Argyll Robertson pupil that could prove my premise that the usual action of the ciliary muscle is responsible for the retained accommodative response of the Argyll Robertson pupil. This new viewpoint is presented merely to show that a diseased and hypoactive iris is compatible with a hyperactive accommodation response.

Confirmation for the supposition that the nerves in the iris in a case of Argyll Robertson syndrome are diseased comes from the work of Orlando and Gambino.[79] They discovered that the colloidal gold curve in the aqueous humor of a paretic patient with the Argyll Robertson syndrome was identical with the curve found in the spinal fluid.

Since the Lange test is subject to experimental error, Orlando and Gambino had six physicians independently perform the tests on the aqueous humor and spinal fluid. All six found the curves to be identical in the two fluids. The paretic colloidal gold curve indicates that there is degeneration and demyelinization in the nerves of the tissue bathed by the fluid.[52, 53]

Conclusions

It is apparent, therefore, that Langworthy's theory of an iridopathogenesis of the Argyll Robertson syndrome is the most tenable current theory. It is consistent with the general pathology of neurosyphilis and with the abnormal irises found in the Argyll Robertson syndrome. It is also consistent with a high incidence of unequal development in the two eyes, a circumstance which results in a unilateral Argyll Robertson pupil. I have shown that the iris pathogenesis theory is compatible with the overactive accommodation response.

Since Langworthy and Ortega postulate a widespread involvement of the nerves of the iris, the apparent anomaly of immobility combined with miosis seems to be a logical consequence of this involvement. They do not explain, however, the mechanism by which the pathologic process in the iris could produce the complete Argyll Robertson syndrome. This mechanism cannot be determined except by adequate histologic examination of the irises of those patients who have had an accurate clinical diagnosis of Argyll Robertson pupil.

In addition, study of the colloidal gold curve of the aqueous humor of normal and of Argyl Robertson pupil eyes should prove fruitful. The hypothesis at present considered most tenable is based on some of the assumptions made by Langworthy and Dupuy-Dutemps, as well as on my application of the Helmholtz theory of accommodation to the Argyll Robertson pupil. This hypothesis is:

The essential and primary pathologic proc-

ess is in the iris. The blood vessels in the iris become diseased, as they do in the dorsal roots and optic nerve in neurosyphilis.

The resulting partial occlusion hinders nourishment to the tissues and causes an atrophy of all the nerves in the iris. The blood vessels, which are normally coiled and very elastic, become rigid. The radial muscle fibers, which normally work mainly by elastic contraction when the sphincter pupillae is inhibited, become atrophied. The sphincter muscle, which is a muscle derived from neural ectoderm, also atrophies.

All these factors result in a pupil that is inactive to all stimuli. The miosis is a static condition induced by inertia of the elastic properties of the blood vessels and radial muscle fibers (dilator muscle).

The reaction to accommodation is the result of the centripetal movement of the whole iris when the ciliary body contracts during accommodation. In the case of the miotic, rigid iris of the Argyll Robertson syndrome, this centripetal movement is not opposed by the normal tone of the structures of the iris. Therefore, the accommodative movement of the atrophied, rigid iris is more effective than it normally is.

The actions of drugs on this diseased iris may be explained as follows:

Atropine produces the partial dilation of the Argyll Robertson pupil solely as a result of the effect of this drug on the ciliary muscle. When the ciliary muscle is paralyzed, the root of the iris is pulled in a direction away from the center of the pupil and partial dilation follows. This assumption is consistent with the new viewpoint that the accommodative constriction of the Argyll Robertson pupil is brought about by a centripetal movement of the root of the iris in accommodation.

Explanation of the action of cocaine and benzedrine in the case of the Argyll Robertson pupil is purely speculative, since the mode of action of these drugs on the normal pupil is not known. These drugs may act by stimulating the sympathetic innervation of the blood vessels. Since the Argyll Robertson pupil is inactive when it is exposed to circulating adrenaline, this supposition can be questioned.

The reaction to light that follows the dilation produced by cocaine and benzedrine suggests that the Argyll Robertson pupil is ordinarily immobile to light because the weakened constrictor muscle cannot contract against the resistance offered by the rigid blood vessels. When these blood vessels are stimulated, however, they become less rigid and the pupil dilates in the dark due to elastic recoil. The sphincter pupillae can then overcome the lowered rigidity.

This theory is based solely on the supposition of an abnormal iris and does not rely on a central nervous-system lesion. Therefore the theory makes it seem worth while to pursue further studies of the iris and aqueous fluid of patients with an Argyll Robertson pupil. These studies will be reported when they are completed.

Summary

1. A review of the experimental work that has been designed to prove a central nervous-system pathogenesis of the Argyll Robertson syndrome has shown that no experimental lesions have produced a permanently miotic pupil that is also rigid to light.

2. A review of the pathologic studies of patients with neurosyphilis has shown that these studies have failed to demonstrate a consistent central nervous-system lesion in any of the areas that serve the dilating and constricting mechanisms of the pupil.

3. A review of reports of the clinical examinations of the iris of patients with an Argyll Robertson pupil has shown that these reports demonstrated that an abnormal iris is present in every case of an Argyll Robertson syndrome. Pathologic study has proved this. Chemical tests of the aqueous humor substantiate these findings.

4. Reference is made to a recent study[105] of 46 patients with neurosyphilis in whom a high incidence of unilateral Argyll Robertson

syndrome was found. This high incidence is compatible with the pathologic process in the iris that accompanies the syndrome but is incompatible with the theories of a central nervous-system pathogenesis of the Argyll Robertson pupil.

5. On the basis of these facts, I present a theory that the abnormal iris blood vessels, nerves, and muscles are responsible for a small, immobile pupil. This theory proposes that the overactive accommodation response of the Argyll Robertson syndrome is a result solely of the forward movement of the root of the iris when the ciliary body contracts. Moreover, I suggest that the partial pupillodilation which follows atropinization of the Argyll Robertson pupil eye is a result of traction on the root of the iris by the concomitant paralysis of the ciliary muscle.

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CLINICAL PATHOLOGIC CONFERENCE

PARKER HEATH, M.D.
Sullivan Harbor, Maine

CASE E52-410

HISTORY

The parents of the patient were healthy. The patient himself, aged 14 years, was the sixth of seven children. His younger sister, aged 10 years, is a mongoloid child. Eleven months before this visit, vision in the patient's left eye was 20/20. Nine months ago spots began to appear before the left eye. Following this, a progressive painless loss of vision occurred. Some flashes of light were seen in the temporal field when in a dark room. Six months before this visit the patient gave a history of being struck with a tennis ball in the left eye.

CLINICAL FINDINGS

The refractive error was not significant. Vision in the right eye was 20/20; left eye, hand movements at one foot. Visual field measurements in the right eye were normal, but could not be measured in the left eye.

Slitlamp examinations showed: R.E., normal; L.E., flare and cells were noted, but no keratic precipitates. The vitreous was hazy and contained yellowish exudates.

Fundus examination of the right eye was completely negative. The fundus of the left eye showed what appeared to be massive retinal detachment with many separate cysts or balloons. Numerous vessels could be seen in the detached retina around the disc. The vitreous was largely filled with a white exudate, particularly in the extreme periphery. Round formations, perhaps collections of cells, were seen. Transillumination was not definite.

COURSE

Two months after the first visit, the patient had attacks of severe pain in his left eye. Intraocular pressure measurements were

80 mm. Hg (Schiøtz). The cornea was steamy; the pupil was dilated; and the lens pushed forward.

Examination of the fundus showed the lower portion of the vitreous cavity to be largely filled with exudation; the disc appeared to be pulled forward; a few large retinal vessels were visible in the upper portion of the fundus. Massive separation of the retina and fixed folds were noted. At the 1-o'clock position near the periphery was a reddish tumor which was thought to be a cyst or a hemangioma of the von Hippel-Lindau type. A sketch would indicate vessels emanating from the obscured nervehead, scattered spots of hemorrhage, and globe-shaped separations of the retina with fixed folds.

X-ray examinations were made of the globe and orbit. No calcifications, bony disturbances, or mass seen. General physical examinations were normal. Examination of the patient's younger mongolian sister revealed optic atrophy in the left eye with convergent strabismus and a vision ability of counting fingers at two feet. The right eye appeared normal.

Treatment

Enucleation of the left eye was advised and accepted. Postoperatively the patient has remained well.

Differential diagnosis

*Discussion by Dr. Joseph LoPresti**

We are presented with a problem of establishing a diagnosis in a unilateral example of mass in the vitreous. As a corollary to this, the patient had glaucoma with pain. A history is given of trauma to the eye some six months before appearance at the hospital, but the patient complained of spots before his vision and had symptomatology prior to the tennis-ball accident.

We can assume that the trouble in the left eye antedated the injury and was without known cause, as indicated by the history. The onset was painless, with a gradual leading up to the final painful stage.

To begin with one would think of an inflammation severe enough to fill the vitreous —possibly a metastatic abscess—as a likely consideration. The absence of keratic precipitates on the back of the cornea, absence of indicated iris changes, and involvement of the posterior chamber, as well as the glaucoma, tend to rule out a severe uveitis in the stage of an endophthalmitis. The long course, gradual progression, and other signs are further evidence against a devastating uveitis.

One could consider some type of tumor, possibly a metastatic growth, possibly a retinoblastoma. However, the patient is too old for retinoblastoma to be likely. The age is more reasonable for the appearance of a tumor of the hemangiomatous type. The massive retinal separations with fixed folds and the presence of hemorrhage and blood vessels suggest such a diagnosis. Especially significant is observation of a reddish mass in the periphery. No family history of similar disease is given and no suggestion of cerebral or cerebellar involvement is indicated. X-ray examinations were said to be negative. It is possible, however, for such a growth to be found in one eye only—the cerebellum free.

An additional diagnosis to consider in the young male is a massive separation of the retina due to hemorrhage with excessive reactivity. This is known by some as Coats's disease. The appearance of the lesion in this case is, however, against this last-mentioned diagnosis.

Another consideration is that of nematode endophthalmitis. We are not furnished with a protocol of any history relating to pets or exposure to the same. A unilateral endophthalmitis of this type is more commonly found in juvenile males. The parasite usually is a nematode. It is quite reasonable to exclude this possibility in this case. The course and progression from the first to the last

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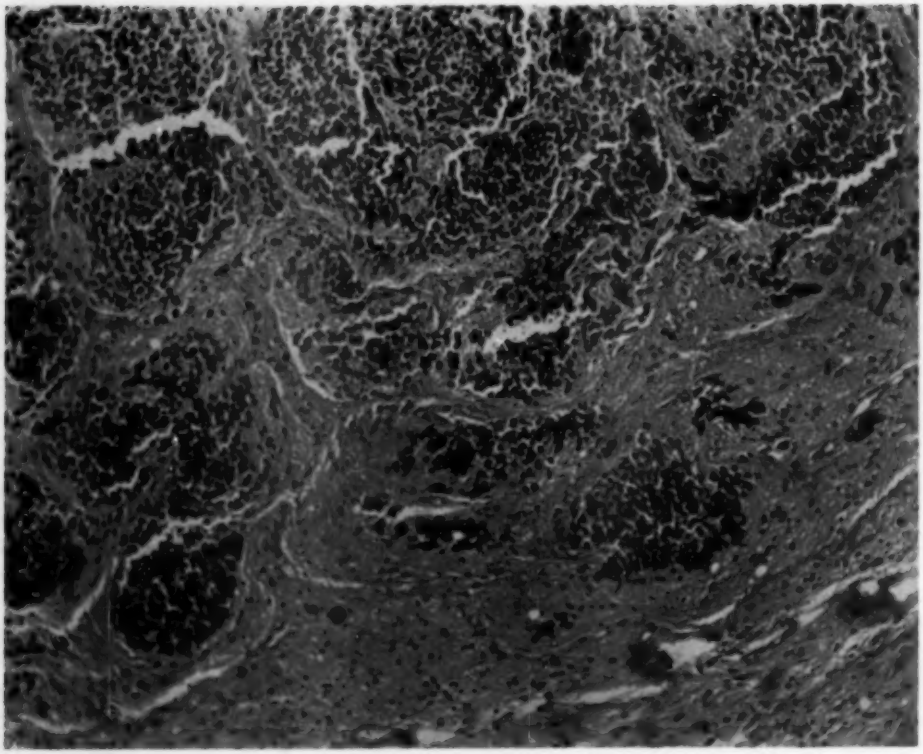

Fig. 1 (Heath). Tumor cells within the cross section of the optic nerve, beyond the lamina cribrosa.

stage would be unlike a nematode endophthalmitis. Glaucoma rarely occurs in parasitic disease but it is possible. The nature of the separation as described would also be somewhat unlike that following parasitic invasion.

I come back, therefore, to the notion that the cause of the unilateral separation in this patient is due to vascular tumor, angiomatosis of the von Hippel-Lindau type.

Clinical diagnosis. Massive separation of the retina. Angiomatosis with Coats's reaction.

Dr. LoPresti's diagnosis. Massive separation of the retina due to vascular tumor—angiomatosis retinae.

Pathologic diagnosis

Retinoblastoma, with extensive intraocular growth, massive invasion and separation of the retina, extensive invasion of the optic nerve beyond the lamina cribrosa.

Comment

Dr. Parker Heath

Dr. LoPresti's analysis of this protocol is consistent with the appearance of the case and the law of averages regarding the tumor. Retinoblastoma at the age of 14 years is very unusual. Of course these tumors have been described in four or five adults. The average age, however lies between two and three years. The discusser did what all the rest of us would have done, namely, after mentioning the possibility of retinoblastoma discarded it as unlikely in the face of the law of averages.

It is interesting that the patient had a mongoloid sister with an eye on the same side exhibiting optic atrophy. This red herring failed to disturb the discusser.

The floating materials described in the vitreous in the protocol no doubt were seeds of the tumor. Advanced stage of the growth is indicated by the glaucoma, large size of growth, and somewhat by the extensive optic-nerve involvement. The latter may occur early.

A note on the management of this case: Following enucleation the patient was given a course of X-ray therapy to the stump and by cross firing. Roentgen and nitrogen-mustard therapy are currently being used.

Ophthalmic Miniature

Retrobulbar Neuritis

"Marie Koch, aged five and one-half years, suddenly lost the vision of both eyes. She had always been well nor had she had the ordinary diseases of childhood.

"The face was pale, the pupils widely dilated, and continual rubbing of the nose suggested intestinal parasites and there was a history of passing ascaris lumbracoides. She awoke one morning, groped her way about while the eyes wandered aimlessly like those of a blind person. . . . The pulse was 160 and weak but the bodily functions were normal. No signs of inflammation of the eyes, the pupils did not react to light, and no signs of increased intraocular pressure. Disc outlines were lightly blurred, intracranial lesions were ruled out; embolism also because of bilaterality. Blood was drawn from the temples, vesicants applied to the neck, large doses of calomel, hot foot baths and mercury rubs were without effect. As the child had been blind for 10 days and because of the arterial anemia of the retina, I determined to create a negative pressure within the eye to induce a return of the circulation as von Graefe had done in cases of embolus of the central artery of the retina. Accordingly I did a broad iridectomy on the right eye and limited myself to a paracentesis of the left eye.

"Twenty hours later, both chambers were re-established without reaction and the eye upon which an iridectomy had been done recognized light and followed the hand movements. The pupil response of the left eye was in sympathy with the stimulous applied to the right eye but it was amblyopic as at first.

"On the following day the child counted fingers at two feet but the left eye was unchanged. I did not delay further but did an iridectomy upon the second eye. The course of this eye was like the first and upon removing the bandage the next day, light perception was evident. Fifteen days later the child could distinguish points one-half mm. apart and the fields were normal. Three days after the operation the disproportion between the retinal arteries and veins had disappeared. I have given the name *Ischaemia Retinae* to the condition, basing it upon the definition offered by Virchow. I am well aware that further proof is necessary to support my views of the case."—Alfred Graefe, Ischaemia Retinae, *Archiv für Ophthalmologie*, **8**:143 (Part 1) 1861.

"Anna P., aged three and one-half years, the daughter of a physician, had always been well except for measles and bronchitis in the first two years of her life and a profuse nasal secretion. After an attack of angina tonsillaris which soon passed off without any suggestion of diphtheria, the nasal secretion ceased. In about three weeks, objects appeared dim and the next day she was unable to go about alone. She complained of a thick smoke before the eyes in which red drops and shining balls moved continuously. The second day she was completely blind. Five days after the symptoms described, my findings were face pale, pulse low, no fever, no light sense, pupils ad max, some nystagmus and light haziness of the papilla borders. Retinal arteries are narrow, veins overfilled and tortuous but no hemorrhages. The therapy consisted of calomel, local mercurial inunction, copper sulphate to the nasal mucous membrane, and rest in a dark room. Eleven days after the complete loss of vision, I performed an iridectomy upon the left eye. I did not operate upon the right eye as I did not care to lose the benefit of a comparison. Three days later to my great joy light perception was shown and, 'O Horrors, the same condition was present in the unoperated eye.' . . . Naturally we find ourselves at this time upon a hypothetical basis and are compelled to find an explanation for this sudden and terrifying loss of vision that will conflict least with known facts. Such appears to me to be the assumption of a retrobulbar neuritis."—Albrecht von Graefe, Concerning neuroretinitis and certain fulminating blindings, *Archiv für Ophthalmologie*, **12**:114, 1866.

Editorial comment. Sudden blindness without ophthalmoscopic evidence was a serious problem until a relatively recent date. The two case reports quoted here show how two excellent clinicians, Alfred Graefe and his nephew, Albrecht von Graefe, interpreted two very similar cases. It illustrates the difficulty of diagnosis in the absence of laboratory findings. We are faced with the same problems today in cases which provide little or no material for the pathologist. Unless the eye is painful or the question of malignancy arises we are often in a quandry. (R.I.L.)

NOTES, CASES, INSTRUMENTS

HORIZONTAL EXCISION OF TARSUS

In the correction of ectropion luxurians

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Philadelphia, Pennsylvania

Spastic ectropion tends to perpetuate itself. The folding at the attached border of the tarsus causes venous strangulation and consequent engorgement. Exposure of the conjunctiva, eversion of the lacrimal punctum, and the constant wiping of the lid by the patient add to the inflammation and maintain the spasm of the orbicularis oculi.

As a result, the exposed conjunctiva may become dry, rough, and thickened. Or, it may become swollen, hyperemic, and productive of an exuberant, irregular growth which resembles granulation tissue, a condition which is called ectropion luxurians or ectropion sarcomatosum. The first term is much to be preferred.[1]

The patient herein presented showed the dry, uniformly thickened type of spastic ectropion in the lower right lid, and an ectropion luxurians in the upper left lid. The correction of the lower lid posed no unusual problem but that of the upper left lid threatened complications if approached by standard methods.

The hyperplastic mass extended horizontally from the external canthus to a point corresponding to the nasal third of the cornea and had a very rough surface. If the lid were reposited without removing all this tissue, the eye would be exposed to trauma and the lid would be extremely thick.

A horizontal excision of the tarsus removed the entire mass and, in combination with a small Kuhnt operation, corrected the ectropion.

Case report

F. T., a Negro, aged 58 years, was first examined in the Out-Patient Department of the Pennsylvania Hospital in August, 1951, complaining of burning and lacrimation of both eyes during the past five years.

There was a uniform ectropion of the entire lower right lid with moderate thickening. The outer two thirds of the upper left lid were ectropic with a large hyperplastic mass on the conjunctival surface, the size of the mass and the extent of the ectropion increasing toward the external canthus (fig. 1). Vision was correctible to 6/6 in both eyes and the fundi were normal.

Since the patient refused surgery the affected lids were painted with glyceride of tannin every second week and ointments of bacitracin, neomycin, and various sulfa compounds alternately prescribed. There was no improvement in the course of two years' treatment. Permission to operate was finally obtained.

On June 15, 1953, under local anesthesia the lower right lid was corrected by a Kuhnt-Szymanowski operation.[2]

Still under local anesthesia the upper left lid was grasped in a large Desmarres lid clamp which isolated the hyperplastic mass, permitted proper control of the lid, and gave an almost bloodless field. While an assistant held the clamp, I excised a horizontal, oval section of tarsus underlying the mass, using a small Bard-Parker knife

Fig. 1 (Gifford). Appearance of patient before operation (June 15, 1953).

Fig. 2 (Gifford). Lines of incision.

(No.-3 handle, No.-15 blade) and taking great care not to injure the muscle and skin beneath. The section was approximately 18-mm. long and six-mm. wide, and left a narrow three-mm. strip of tarsus adjoining the lid margin (fig. 2). The apposing tarsal edges were joined with black-silk sutures.

The ectropion was almost, but not quite, corrected by this procedure. Accordingly, the inner third of the lid was split by moving the clamp to that position and cutting along the lid margin with the Bard-Parker knife. A small triangular piece of tarsus was excised and the tarsal margins sutured according to method of Kuhnt.[2] The lid was now in good position. A pressure bandage was applied to both eyes.

The pressure bandage was changed and the eyes dressed daily for a week. Then bandage and sutures were removed. Except for a moderate amount of tarsal edema the appearance was good. The method used in the upper left lid had created two folds of surplus skin; a long horizontal one over the outer two thirds of the lid, and a short vertical one over the inner third. By the time the pressure bandage was removed the lid was entirely flat.

Fig. 3 (Gifford). Appearance of patient after operation (June 26, 1953).

On June 26th, the tarsal edema had subsided, the eyes were quite comfortable, and the lids were in good position though they remained a little thickened (fig. 3). When last seen on September 21st, this thickening had decreased.

Conclusion

Ectropion luxurians is not satisfactorily treated by simply correcting the ectropion. The rough, irregular mass of tissue which proliferates upon the conjunctival surface of the lid cannot safely be allowed to impinge upon the cornea. In addition such a lid, when in normal position, would bulge forward in an unsightly fashion. These considerations are particularly true when the upper lid is involved.

In this case a horizontal excision of the mass with the underlying tarsus, and suturing the tarsal edges, at once removed the offending growth and went far toward correcting the ectropion. A small Kuhnt operation completed the correction. It must be kept in mind that the upper tarsal plate is only 11-mm. wide at its greatest width and it would seem unwise to remove a section measuring much more than half this width.

1913 Spruce Street (3).

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MALIGNANT MELANOMA OF THE UPPER CONJUNCTIVAL FORNIX

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Malignant melanomas of the conjunctiva are extremely rare. Because of the unusual localization, the advanced stage of the malignancy, and the relatively long survival in spite of failure of radical operation, it seems desirable to publish the following case.

CASE REPORT

History. E. N., a white woman, aged 70 years, came to this office for the first time on November 30, 1948. About one and a half years previously she noticed a swelling over the right eye, which slowly grew in size and, within the last few months, developed into a conspicuous fleshy growth protruding between the lids. Because of religious beliefs, she refused to accept medical aid.

Examination. A huge reddish, lobulated growth covered the entire right orbital entrance. Hidden behind the tumor, neither lids nor eyeball could be seen. The tumor pressed against the bony margin of the orbit. On pressing or lifting, the highly vascularized surface of the growth bled easily. The origin of the tumor could not be established. A choroidal tumor with extraocular extension, appearing as an epibulbar growth, was suspected. The face, especially on the right side, the right upper lid, the neck, and chest showed several small pigmented nevi. The left eye showed no pathologic condition.

Course. The patient was hospitalized and a histologic examination of biopsied material in frozen section (Dr. A. R. Camero) revealed the presence of a malignant melanoma; the diagnosis was later confirmed by examination of the excised tumor. Orbital exenteration was recommended but the patient refused and, with the consent of the Tumor Board of the hospital, it was agreed to remove only the unsightly portion of the growth.

On the operating table it became evident that the growth originated not from the globe but from about the middle of the upper conjunctival fornix. It was attached to a five-mm. base by a very short peduncle, so short indeed that the tumor was being pulled back against the orbital entrance. The tumor was cut off at the base of the peduncle and the base itself was coagulated about two to three-mm. deep. The upper lid was in a reverted position, pressed against and under

Fig. 1 (Schaeffer). Appearance of patient before first operation in 1948.

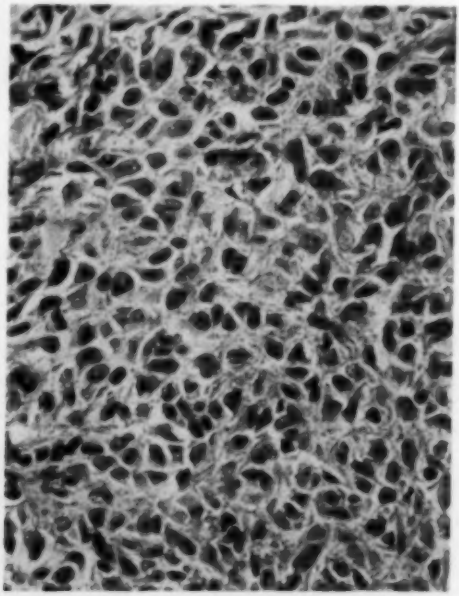

Fig. 2 (Schaeffer). Microscopic section after first operation.

the upper orbital margin; the eyeball was pressed down, simulating enophthalmos.

In a few days both the upper lid and the globe regained their original positions. Further exploration revealed a completely normal eye without an apparent pathologic condition. Vision, with correction (+4.0D. sph. ⊃ +1.75D. cyl. ax. 105°), was 20/20. The visual fields were normal. Ophthalmoscopic examination, transillumination, and radiography have shown no signs of global or orbital extension of the growth.

Recurrence. The patient was expressly asked to come back at short intervals for check ups but she did not do so until January, 1953. The orbital content, covered by the bulging upper lid, was now extruding, the seemingly atrophic eyeball, surrounded by highly vascularized edematous tissue, was immobilized and pressed forward and down against the lower orbital margin.

The globe itself was blind and probably disorganized. The cornea was vascularized and leukomatous. Behind the globe, a dark mass, not clearly visible, suggested the presence of a destructive growth. The skin of the upper lid showed a blackish discoloration and pigmentation formed a sharp line at the margin of the lower lid.

The tumor filled the orbital cavity; no movement of the eye could be elicited. No pathologic process was found in the left eye.

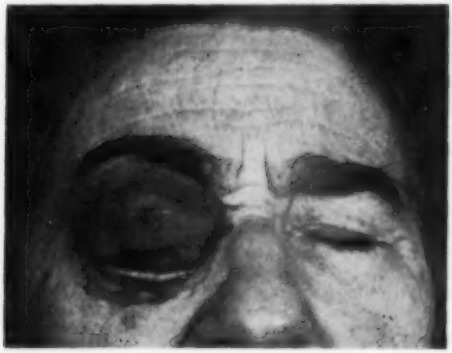

Fig. 3 (Schaeffer). Appearance of patient before second operation in 1953.

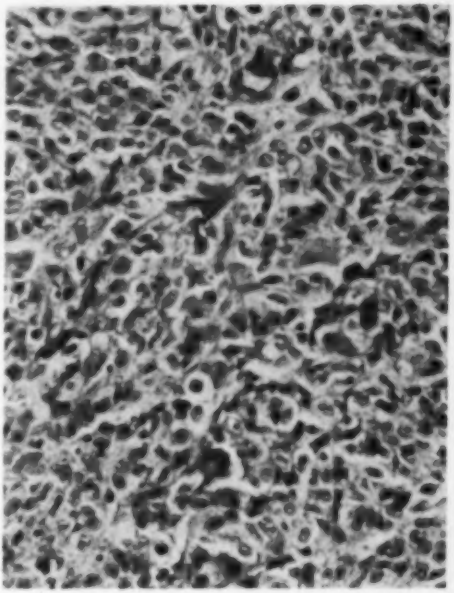

Fig. 4 (Schaeffer). Microscopic section after second operation.

Because of the advanced stage of the malignancy, radical surgery was advised. On March 11, 1953, Dr. S. L. Perzik performed an exenteration of the orbit from which the patient did not recover. After the operation she remained unconscious and died on the third day, probably due to cerebral hemorrhage. Autopsy permission could not be obtained. Again the excised specimen was diagnosed as extraocular malignant melanoma.

Comments

Merigo de Treigny mentions that Lyder found eight conjunctival melanomas among 45,000 clinical cases; Morax found only half this number. Licsko found eight cases among 22,000. Reese, in reviewing the literature on this subject in 1941, found 42 cases "which could be identified either definitely or presumably" as cancerous melanosis.

Most of such tumors are localized at the limbus, or at the bulbar conjunctiva, lacrimal caruncle, or semilunar fold. Malignant mel-

anosis originating at the palpebral conjunctiva is an extremely rare finding (Trapesontzeff, Scalzitti, Posey, and Triossi). I have found only two cases in which the tumor originated in the cul-de-sac: a pedunculated tumor described by Roemer, another on the upper conjunctival fornix described by Rifa. If one in every 100 malignant melanomas of the conjunctiva arises at the fornix, the case herein reported shows an incidence of one in 4,000,000 clinical eye cases.

There was no way to establish whether in my case the growth developed from a nevus or was a primary melanoma of the conjunctiva proper.

Mawas denied the existence of primary melanotic growths and stated that most of these tumors were fundamentally, and always, nevus epitheliomas.

Reese's interpretation is diametrically opposite. From his 42 cases already mentioned only two "could be definitely identified as malignant melanoma which arose from a pre-existing nevus." Reese believes that cancerous melanosis of the conjunctiva and skin of the lids is 10 to 15 times more frequent than malignant melanoma of nevus origin.

The presence of skin nevi in my patient does not prove that the melanotic tumor of the fornix originated from a nevus.

Another interesting feature in the case herein reported was the advanced stage of the disease for, in spite of the fact that, at the time of the first operation the growth was already of considerable proportions and, furthermore, in spite of her refusal of radical surgery, the patient survived nearly five years. This would be a satisfactory outcome under the most favorable conditions.

A similar pedunculated tumor at the limbus was described by Fernando. In his case also, the patient refused radical operation and the tumor was simply excised. The growth did not recur locally but widespread metastases caused death within two years.

There was no evidence of hereditary factors in the present case.

Summary and Conclusion

1. A case of primary malignant melanoma of the upper conjunctival fornix is reported. This is considered to be of rare occurrence.

2. The patient survived nearly five years without radical operation.

6317 Wilshire Boulevard (48).

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CHOROIDITIS WITH LYMPHOCYTIC CHORIOMENINGITIS

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After we had seen a patient with lymphocytic choriomeningitis, whose principal symptom was bilateral chorioditis, the literature was reviewed but no case of choroiditis in this condition could be found.

Wenner, Swann and Heilbrunn have reported[1] a case with absent pupil reflex. A case with homonymous hemianopia and loss of conjugate deviation has been reported.[2] Another case with diplopia and photophobia is recorded.[3] Finally, the literature describes a case with photophobia and papilledema.

CASE REPORT

A 31-year-old white man, whose present illness started on January 27, 1953, with malaise and fever, noticed, five days after onset, a loss of vision. He was put on penicillin and other antibiotics for three days with no improvement.

The patient was seen by one of us (H. F. J.) on February 6, 1953, and findings at that time were:

Vision: Light perception, O.U., not improved with lenses. There was a dense vitreous clouding and the fundus details could not be clearly seen.

He was admitted to the Easton Hospital on February 6, 1953. The temperature was 101°F. Except for bilateral acute choroiditis, no definite physical findings were present.

Laboratory report: R.B.C., 3,950,000; W.B.C., 10,050; hemoglobin, 11.0; Wassermann, negative; chest plate, negative; skull and sinus plate, negative. Spinal fluid: Protein, 5 mg. percent; sugar, normal; cell count —58 cells per. cu. mm.; 96 percent, lymphocytes; four percent polymorphonuclears.

The patient was given 15 million units of typhoid vaccine on admission and one-percent atropine (one drop in each eye, twice daily).

On February 7, 1953, he was started on cortisone (50 mg., four times daily). The differential diagnoses on admission were: (1) Poliomyelitis, (2) coxochkie virus, (3) mumps encephalitis, (4) lymphocytic choriomeningitis.

On the basis of the spinal-fluid findings and the clinical course since January 22, 1953, we felt that lymphocytic choriomeningitis was the most likely diagnosis. Unfortunately, our laboratory was not equipped to carry out serologic studies for the disease. Treatment with cortisone was initiated even though laboratory confirmation was not available.

Because of patient's severe loss of vision and malaise, serologic samples were sent to the U. S. Public Health Service, after treatment with cortisone. The serologic report during the period of convalescence was negative. Cortisone may reverse the serology of virus encephalitis; cortisone does reduce lymphocytic elements.

By February 15, 1953, the vitreous haze had cleared and vision had returned to 20/50 in each eye. Spinal-fluid examination on February 16, 1953, showed that the cells had dropped to six cu. mm. with 90-percent lymphocytes and normal chemical findings.

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HARADA'S DISEASE*

A CASE REPORT

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The complete syndrome of Harada[1] occurs infrequently. It is comprised of bilateral uveitis, bilateral retinal separation, and symptoms of meningeal irritation. Dysacousia, lymphocytosis of the spinal fluid, vitiligo, and poliosis may also be present. In the following case all essential features of the syndrome were present. Special studies were used in an attempt to determine the etiology.

CASE REPORT

History. A 36-year-old, married Negress, in December, 1952, suffered from fever, malaise, emesis, and abdominal cramps. A local physician gave her four intramuscular injections of pencillin. In January, 1953, she had severe diffuse headaches, shortly followed by sudden bilateral decrease in vision. At this time she stopped nursing her one-year-old baby. Ten days following the sudden decrease in vision she entered the University of Nebraska Hospital.

Physical examination showed these positive findings: Small galactoceles of the right breast and an intrauterine pregnancy of two to three months' duration. Vision was limited to counting fingers at one foot in both eyes. External examination of the eyes showed moderate conjunctival edema and slight ciliary congestion. The pupils were dilated by mydriatics previously instilled. Tension was 7.0 mm. Hg (Schiøtz), O.U. Motility was normal.

Ophthalmoscopic examination revealed fine vitreous dust and blurred discs in each eye. The retina surrounding the discs was edematous to the extent of four diopters. In both eyes there was a large bullous detachment below. Scattered through the right fundus were grayish, slightly elevated areas.

Slitlamp examination of each eye revealed an increase in the visibility of the aqueous beam, many cells in the anterior chamber, numerous deposits on the anterior capsule, and beginning posterior synechias.

Course. During the entire hospitalization the patient was afebrile. She received local cortisone (2.5-percent solution) to both eyes. On the 10th day cortisone was started systemically, 200 mg. daily. Because the patient's condition became worse, the dose of systemic cortisone was gradually reduced and discontinued after a total of 12 days.

At this time vision had decreased to shadows in each eye. In both eyes the vitreous was more hazy and the size of the bullous detachments increased. Slitlamp examination revealed small pigment granules on the anterior capsules, a network of fibrin in the anterior chambers, and small corneal precipitates.

Other treatment consisted of 60 gr. of sodium salicylate daily for three weeks, three intramuscular injections of 300,000 units of penicillin, four gm. of Gantrisin (Sulfisoxazole) daily for 10 days, and one intramuscular injection of two cc. of immune human globulin.

Six weeks after admission the patient was discharged. At this time examination showed no retinal detachments. The fundi revealed deep pigmented areas which were more marked in the inferior portions. The chambers were clear of fibrin but showed slight flare. Vision had improved to ability to count fingers with both eyes.

Two months after discharge the vision was 20/60 in the right eye and 20/400 in the left eye. Tactile tension was normal. Peripheral limits of visual fields were grossly normal. The vitreous showed slight haze. The fundi showed areas of depigmentation involving both macular areas. The discs and vessels were normal. Anterior chambers were clear and there were a few "ghost" keratic

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precipitates and marked posterior synechias in each eye. On the head there was one small area of alopecia. Poliosis of the eye lashes was also present.

Seven months after discharge, examination was essentially the same as on the previous occasion with the additional finding of areas of vitiligo on both shoulders. One week prior to this last examination the patient had delivered a normal, full-term boy.

Examinations and special tests in the hospital. Repeated urinalyses gave essentially normal results. Hemoglobin concentration was 11.9 gm. There were 6,700 leukocytes with a differential count of 44 segmented forms, 8 staffs, 17 eosinophils, 1 basophil, 29 lymphocytes, and 1 monocyte. A subsequent differential count two weeks after admission showed 51 segmented forms, 12 staffs, 1 eosinophil, 26 lymphocytes, and 10 monocytes. Corrected sedimentation rate on admission was 31 mm. per hour. Blood serology was negative.

Agglutination tests for typhoid O and H, paratyphoid A and B, Brucella abortus, and proteus OX19 were negative. Complement-fixation tests were negative for Western equine encephalomyelitis, St. Louis encephalitis, lymphocytic choriomeningitis, lymphogranuloma venereum, and Q fever. No serologic evidence of infection with influenza virus, types A or B, was found. On one occasion a methylene-blue dye test for toxoplasmosis showed a titer of 1:64. When repeated on a second sample of serum six weeks later, the titer was negative.

Blood chemistry values, including those for proteins, fasting glucose, cholesterol, and nonprotein nitrogen, were not remarkable. Thymol turbidity liver function test was 4.7 units and cephalin cholesterol flocculation test was negative after 48 hours. The basal metabolism rate was +16 percent.

The following skin tests were negative: first strength and second strength tuberculin (purified protein derivative), Frei, histoplasmin, and coccidioidin.

A spinal tap on admission showed normal dynamics. There was a pleocytosis of 30 cells, all lymphocytes. Total proteins were 78 mg. percent. The Eagle test was negative. The colloidal gold curve was 1234443210. No pathogenic organisms were found on smear or culture.

A second spinal fluid examination performed five days after admission showed 132 cells, 96 percent lymphocytes and four percent polys. No pathogenic organisms were found on smear or culture. Colloidal gold curve was 1111111000. The Eagle test was negative. A third spinal fluid showed a pleocytosis of 42 cells. A blood culture on admission was negative.

Roentgenograms of the skull, chest, and teeth were negative. An electrocardiogram was normal. An electro-encephalogram showed alpha rhythm low voltage, 10 cycles per second, moderately asymmetrical with lower voltages on the left. There were no slow waves. There were short bursts of very low to moderate voltage 35 cycles per second activity over the frontal lobes. This was interpreted as a slightly abnormal fast record suggestive of mild brain damage or irritative process.

An audiogram showed a 20-decibel loss bilaterally at frequency of 1,000 and a 50-decibel loss bilaterally at a frequency of 8,000.

Patient's cerebrospinal fluid was injected in 0.1 cc. doses into the vitreous of the left eye of four rabbits. Spinal fluid from the first puncture was used in the first two rabbits and fluid from the second puncture was used in the second pair of rabbits. There was slight inflammatory reaction in the inoculated eyes for several days following injection. An incomplete cataract formed in two of the inoculated eyes on the second day. Daily examinations of the fundi showed no apparent abnormalities.

One injected eye was enucleated after two weeks, two were removed after three weeks, and the fourth was removed after four weeks. All four eyes were studied microscopically and showed no evidence of in-

flammation. Two of the eyes showed cataractous lens changes and slight retinal damage which occurred from the trauma of the injections.

Adult white mice were inoculated intracerebrally with 0.55 cc. of patient's spinal fluid in an attempt to demonstrate Toxoplasma. No Toxoplasma were found.

Comment

The history of Harada's disease was recently reviewed by Cowper.[2] In his opinion Harada's disease and Vogt-Koyanagi syndrome are both manifestations of the same disease; furthermore, Cowper has proposed a new name, uveo-encephalitis, to include both clinical entities. Hogan[3] also states that it is not certain that these two conditions should be considered as separate entities. Malatesta[4] in a recent case report also stresses the relationship between Harada's and Vogt-Koyanagi syndromes. The case reported here shows clinical features of both entities and is characteristic of uveo-encephalitis.

The etiology of this case was not determined after extensive studies. Attempts were made to demonstrate a virus as an etiologic factor. Previous studies by Takahashi,[5] Tagami,[6] Malbran and Muhlmann,[7] and more recently by Sugiura[8] have strongly suggested a virus etiology.

42nd and Dewey.

We wish to acknowledge the assistance of Dr. Harold Gifford, on whose service this patient was admitted. Appreciation is also expressed to the Communicable Disease Center, USPH Service, Chamblee, Georgia, for performing the complement-fixation tests and methylene-blue dye test.

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Ophthalmic Miniature

I quite agree with Dr. Gowers that it is very desirable that all students should be taught the use of the ophthalmoscope, but, having said this, I may admit at once that I much doubt whether it is possible for those engaged in miscellaneous practice to keep up such a knowledge of the instrument and its revelations as to be able to put it to practical use. Excepting to those who have daily opportunities of using it, there is possibly more risk of error than prospect of useful discovery.

Jonathan Hutchinson,
Archives of Surgery, **1**:279, 1890.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

April 6, 1953

DR. ABRAHAM L. KORNZWEIG, *President*

HISTORICAL NOTES ON THE EYE IN OLD AGE

DR. FREDERICK D. ZEMAN presented a paper on this subject during the instruction period.

ROUND-TABLE CONFERENCE

EYE PROBLEMS IN THE AGED

DR. ARTHUR J. BEDELL, DR. HERMAN ELWYN, DR. DANIEL KRAVITZ, and DR. JOHN M. MCLEAN comprised the panel of discussors; DR. ABRAHAM L. KORNZWEIG acted as moderator.

1. *Can you suggest a treatment for senile macular degeneration; for Kuhnt-Junius macular degeneration? Has endocrine therapy been of any value?*

DR. BEDELL said that the question suggests a difference between the two where none exists. When Junius and Kuhnt published their excellent monograph in 1926, they neither claimed priority of description nor suggested that their names be associated with their review of the literature and the report of several cases with colored and black and white drawings. They were fortunate in having competent artists and their work has been the source of many quotations but the conditions depicted are those which we know as phases of senile macular degeneration.

We should consider the two common forms of senile macular degeneration. The first evidences of one are perimacular flat, deep retinal, or superficial choroidal hemorrhages which enlarge and vary in form with exudates appearing near the center of the extravasation. The exudates and hemorrhages increase until finally, often after years, and after having passed through the circinate phase, the hemorrhages cease and the exudate becomes an organized scar.

In the other type of senile macular degeneration, vessel sclerosis and macular edema and hemorrhages are less conspicuous but often recur until the terminal stage when there is an almost flat macular region with gross choroidal vessel sclerosis.

The aim of treatment should be directed toward the prevention of the vessel breaks and the vessel sclerosis; in other words, stop the advancing changes of age. Unfortunately, Dr. Bedell said he does not have the key to unlock the mysteries of senility. The only drug that seems to slow the process is potassium iodide but, as in all chronic diseases, periods of less rapid advancement may occur and if they do while taking the potassium false conclusions may be drawn.

The results following endocrine therapy have not been encouraging.

Dr. Kravitz asked Dr. Bedell what he thinks of microwave diathermy. Dr. Bedell said he does not believe it has any influence. Dr. Elwyn said that he knows no therapy for any macular degeneration. Dr. McLean also said he does not know of any therapy. Dr. Kornzweig asked Dr. Zeman whether medicine did any good. Dr. Zeman didn't know of anything specific in these cases.

2. *Is there an essential decrease in central vision in old age without visible disturbance in the media or macula?*

DR. KRAVITZ said that, in his experience, there is no depreciation in vision of the aged without demonstrable changes in the media or macula. In advanced sclerosis of the lens, there is more interference with the transmis-

sion of light and there may be a slight diminution of vision especially for small print. Whenever there is subnormal vision without demonstrable abnormalities, a careful history or a previous record will reveal that subnormal vision existed previously.

Dr. Kornzweig quoted from an article by Fisher that 50 percent of old people have decreased vision without findings. Dr. McLean said that there is always a reason for loss of vision although the cause is not always seen. Dr. Bedell said that, with improved ophthalmoscopic methods, we will always find changes which could account for loss of vision. Dr. Elwyn said that he has seen diminution of vision with no pathologic process evident in media or fundi.

3. *How can one differentiate between drusen of the macula and macular degeneration by ophthalmoscopic examination or other means?*

Dr. McLean registered a mild protest about the use of the word "drusen"—he prefers to call them "colloid bodies." It is not always possible to differentiate between the two—ophthalmoscopically; pathologically one can. They sometimes do not disturb the vision, even in younger people.

Ophthalmoscopically, the benign ones are small and smooth, others are irregular and large.

Dr. Kravitz asked about the use of red-free light. Dr. McLean answered that it has no value. The crux of the matter is if there is loss of visual function.

Dr. Bedell said that one can distinguish between layer types and benign forms; he suggested use of red light. Benign lesions progress to macular degeneration over a period of years. Dr. Kornzweig advised the use of red light.

4. *Is there a true histologic correlation between sclerosis of the retinal arterioles and of similar vessels in the brain, heart, kidneys, and extremities?*

Dr. Elwyn said that, in cases of long-standing hypertension, arteriosclerosis occurs in the retinal arteries. It occurs to a greater extent in the arteries of the brain and is most marked in the arteries of the heart, but its extent there stands in no relation to that in the arteries of the brain, the retina, or the kidneys.

Arteriolosclerosis occurs in long-standing cases of hypertension—least in the retina, more in the brain, most in the kidneys. It does not occur in the heart.

Dr. Kravitz said that Dr. Elwyn's comments were not in agreement with work done at the Mayo Clinic. In a large series of cases, it was found less marked in the kidneys than in the retinal vessels. Vascular sclerosis is a late manifestation of kidney disease in Kimmelstiel-Wilson disease.

Dr. Elwyn said that the question of arteriolar sclerosis has been settled a long time; it occurs in hypertension mostly in the kidneys. Biopsy of the kidneys are of little value.

Dr. Kravitz remarked that he only knows what he reads—the reliability of the biopsy was discussed at the Mayo Foundation and they felt that they found it. Dr. Zeman said that there is still some confusion as to the interrelationship. The findings in retina at post mortem have not been studied too well. Dr. Elwyn said that there is a great deal of literature and that correlations have been found.

5. *What is the present status of treatment of venous and arterial occlusion in the fundus?*

Dr. Bedell said that there is much disagreement among clinicians regarding the treatment of retinal vein occlusion. When a branch is closed, nature bypasses the plug by the formation of compensatory vessels. He said that he has not seen this orderly process altered by local or general treatment.

The most diverse opinions are expressed in total vein obstructions where some advocate the use of heparin, dicumarol, and even rutin, while others fail to see any benefit from them.

Dr. Bedell said that he is convinced that, when the vein is totally closed, no drug can reopen it. He said that we must recognize the three ways in which central vein closure terminates without specific treatment. Some patients have complete resolution, another group have organization of the retina, and the third develop hemorrhagic glaucoma. Dr. Bedell said he believes that final judgment should be reserved until the proponents of a given medical treatment can produce photographs of several cases before, after, and during treatment.

He said that there is no worth-while treatment for embolism. It must be remembered that no photographic proof before and after special treatments has been presented. The diagnosis between artery spasms and occlusions calls for very exact examinations and he questioned the diagnosis of embolism whenever the vision is entirely restored. This does not mean that branch embolisms fail to show an improvement but there is never a total cure. The empiric treatment should be rest, the reduction of high blood pressure, if present, the care of cardiac symptoms, and the lessening of the apprehension regarding the likelihood of a similar catastrophe in the other eye.

The users of vasodilators have not demonstrated any startling results and the drugs for retinal vein closure have also failed. There are, however, several excellent clinicians who place their faith in both types of medication.

In complete vein occlusion, a mydriatic should never be used. Dr. Bedell said that he usually gives a weak eserine solution (gr. 1/30; oz. 1.0) once a day in the hope that glaucoma may be prevented.

Dr. Kornzweig asked about the use of trypsin. Dr. Bedell said that he had no experience with this drug. Dr. Elwyn said that embolism can only occur in bacterial endocarditis. As for venous occlusion, he saw no therapy for it. Incomplete obstruction can recover in a day or two.

Dr. Kravitz agreed with Dr. Bedell that recovery is possible in spastic carditis. In venous closure he said he has seen almost complete closure which did well, with dicumarol and heparin therapy.

Dr. McLean said that there are some therapeutic measures in both cases. A central venous occlusion may not be total. If a thrombus can be prevented from building up, some good results can be obtained with prompt and energetic anticoagulant therapy. As for arterial occlusion, the use of prompt measures, such as vasodilators, and mechanical procedures, such as paracentesis, anoxia, and also diminished air pressure, can give good results.

Dr. Bedell said that some cases have good results without any therapy. He said that he never saw any effect from paracentesis or massage.

6. Has there been any noticeable improvement in retinopathy due to hypertension or diabetes from the use of antipressor drugs or vasodilators?

Dr. Kravitz said that none of the presently known drugs can favorably influence the retinopathy in diabetes. The process is accelerated when vascular sclerosis supervenes. In hypertension, with vascular sclerosis and retinopathy but without vasospasm, the action of dilators or antipressor drugs is too fleeting to be of any benefit. If, however, these are combined with other measures—that is, operation, loss of weight where indicated, mental relaxation, periods of rest—to bring down the blood pressure more or less permanently, there can be considerable clearing up of the retinopathy.

When vasospasm is an additional prominent finding, then vasodilators and antipressor drugs, by relaxing the blood vessels and improving the nutrition of the retina, can result in absorption of the existing exudates and a lessening of the hemorrhages. This was shown to be so by Nicholls (Am. J. Ophth., **35**:1753, Dec., 1952) who treated a series of cases of central retinal edema with vasodilators. He concluded that the greater

the vascular sclerosis present, the poorer the result; the more that vasospasm predominates, the better the result.

In hypertension secondary to kidney disease, the retinal vascular sclerosis is a late manifestation of the disease and cannot be influenced by vasodilators except temporarily when vasospasm is present.

Dr. Elwyn said that the question was not clear. He has seen some effect with use of priscoline—when the blood pressure was markedly reduced, the fundus changes disappeared. Dr. Bedell said that the transitory exudate and hemorrhages may disappear but the vessels do not change. The Smithwick operation is being done less and less because it does not benefit the patient too much.

7. *Does tearing in the aged present a special problem? Discuss dacryocystectomy versus dacryocystorhinostomy.*

Dr. McLean said that tearing does not present any special problems in the aged. In regard to dacryocystectomy and dacryocystorhinostomy, there is still a place for cystectomy when the sac has a tumor or mucocele, otherwise he advised a rhinostomy.

The age of the patient is not a contraindication to surgery. Dr. Bedell said that, if the patient is very old, one should not have rhinostomy performed. Dr. McLean asked, why not?

8. *Is keratitis sicca a problem of aged patients? Is it one that may be overlooked because of failure to do testing for tears?*

Dr. Elwyn replied that he had not seen keratitis sicca in the last few years but perhaps he had overlooked it. Dr. Bedell remarked that it usually occurs in the middle-age group. Dr. Kravitz said that he had only seen one case. Dr. McLean said that the majority of cases occurred during the menopause and especially in women.

Dr. Kornzweig said that it is more common than usually thought to be. It is often overlooked, and a tearing test should be done more frequently. In the aged, the lacrimal gland diminishes secretion. Schimer's test and rose-Bengal tests were mentioned.

In discussion, Dr. F. H. Theodore mentioned that hypothyroidism, occurring in older people, was an often unappreciated cause of bilateral epiphora. Ocular examination of these patients revealed no abnormalities of the conjunctiva or lacrimal passages whatsoever. In a number of such cases treated by him, administration of thyroid caused the tearing to stop dramatically. While the fact that hypothyroidism could cause tearing, probably as a result of excessive formation of tears, was not unknown to internists, most ophthalmic texts did not mention it. The only reference he had noted concerning tearing in hypothyroidism was by Valiere-Vialeix in the *Traite D'Ophtalmologie.*

9. *Is there any special attention that should be given to the aged patient after an operation on the eye?*

Dr. Bedell said that the prime consideration in the treatment of the elderly is the stabilization of the circulation by an early shift from the recumbent to the sitting position, dangling of the legs and getting out of bed. Circulatory derangement may become manifest, with mental aberration and, not infrequently, complete disorientation.

The best way to calm the excited patient is to remove the dressing from the unoperated eye and get him out of bed. If this fails, he should be sent home where he almost invariably promptly recovers his mental equilibrium and usually the eye heals satisfactorily. The ordinary sedatives such as the barbiturates are rarely of benefit.

Dr. McLean said that one should not bandage the unoperated eye, and also suggested the use of a few kind words from time to time. Dr. Kravitz endorsed Dr. Bedell's suggestion to get the patient home early.

10. *Outline the postoperative care of aged patients with retinal detachment.*

Dr. Kravitz said that it is fortunate that

simple retinal detachment is not too frequent in the aged. The oldest patient he had operated was 67 years of age, and retinal detachment followed a cataract extraction in that case. As with cataract extraction, the treatment is individual; with some patients, the regular conservative treatment can be used. However, if necessary, he said he would not hesitate to let the patient out of bed on the second or third day.

A light pressure bandage over both eyes would immobilize the eyes and if the unoperated eye has to remain open, a light pressure bandage over the operated eye should be applied. If the detachment is in the upper part and bedrest is essential, then frequent passive exercise of the limbs with frequent alcohol rubs should be instituted.

If vision is serviceable in the other eye, then surgery should be avoided; however, if there is no serviceable vision in the other eye, then one will have to do the best one can.

Dr. Elwyn said that he doesn't know when old age begins and that he treats all patients alike. Dr. McLean said he had nothing particular to add. Dr. Kornzweig added that he had two cases of retinal detachment, one patient remained in bed for four weeks; one patient remained in bed for one week, results in both cases were poor.

11. *Does glaucoma in the aged show characteristics different from those of younger individuals? What is your attitude toward early surgery in the older patient?*

Dr. McLean answered that it is not glaucoma which differs but the patients are different. Glaucoma starts late in life and runs a slow course. The older patients should be treated conservatively in accordance with their intraocular pressure, visual fields, and life expectancy. In younger patients, early surgery is the key.

Dr. Kravitz seconded Dr. McLean's comments. Dr. Bedell and Dr. Elwyn both agreed.

12. *Would you ever operate on both eyes of an aged patient at the same time, for either glaucoma or cataract?*

Dr. Elwyn replied that, in acute congestive glaucoma where the tension cannot be lowered by other means, he would operate on both eyes at the same time. He said he would not operate on both eyes at the same time in chronic simple glaucoma or for the removal of cataracts.

Dr. Kornzweig cited a case in which he had to operate for glaucoma in both eyes; there were no complications.

13. *What type of operation would you advise for a patient with a secondary glaucoma due to a swollen lens?*

Dr. Bedell said that he would operate under general pentothal anesthesia and do a broad conjunctival flap with a complete marginal iridectomy and would remove the lens with an erisophake.

Dr. McLean said that he uses local anesthesia and forceps. The size of the conjunctival flap makes no difference. He suggested use of sutures and perhaps even a peripheral iridectomy, and use of a posterior sclerectomy if tension is not reduced.

Dr. Kravitz said he uses local anesthesia, an erisophake, and full iridectomy. Dr. McLean asked what a full iridectomy contributes. Dr. Bedell replied that patients do better with it. Dr. Kravitz felt that the iris tends to prolapse and for that reason he does a full iridectomy.

Dr. Kornzweig remarked that, apparently, no one had suggested use of a glaucoma operation first.

14. *Is there any way to determine whether primary optic atrophy in the aged, with low or normal intraocular pressure, is due to glaucoma or other causes?*

Dr. Kravitz said that, at times, it is difficult to differentiate between a simple atrophic excavation of the disc and a true glaucomatous excavation.

A typical glaucoma field would favor diagnosis of glaucoma as against a concentric

contraction which would favor the diagnosis of a primary optic atrophy. A disturbance of the central field or loss of central color perception would favor diagnosis of a primary optic atrophy. X-ray studies would rule out sclerosis of the internal carotid arteries in which such a sclerosis causes glaucomatous excavations with typical glaucoma fields.

The presence of marked vascular sclerosis with disappearance of the smaller vessels would favor diagnosis of an optic atrophy secondary to loss of nutrition. Lastly, taking the tension frequently for 24 hours might reveal a period of increased intraocular pressure and so establish the diagnosis.

Dr. Elwyn reported a case of so-called glaucoma with cupping. There was no increase in intraocular pressure. It was not glaucoma but one of the cases of cupping reported by Dr. Knapp.

Dr. McLean reported a case that has had at least two attacks. He said that a study of the aqueous flow would be of great help.

Dr. Bedell said that one of the 10 cases reported by Dr. Knapp was his. He said one should not make a diagnosis of glaucoma on excavation alone.

Dr. Kornzweig asked whether there was such a thing as low-tension glaucoma. Dr. Kravitz answered: what else would you call it? Maybe observation over 24 hours would help. Also, he mentioned, measuring the outflow might be the answer.

15. *What extra precautions are necessary in patients with corneal dystrophy requiring cataract extraction or other intraocular surgery?*

Dr. McLean replied that this would depend on the dystrophy. In Fuchs' dystrophy there is one definite precaution to be followed, and that is not to use a corneal or limbal incision. In other dystrophies there is no problem. Dr. Kornzweig suggested use of an incision behind the limbus. Dr. Kravitz said that a scleral incision results in greater astigmatism.

16. *Is there any way to determine in advance that the corneal section for cataract would fail to heal after two weeks? Would you prefer silk or catgut sutures in such patients?*

Dr. Elwyn replied that there is no way to determine in advance. In such patients, Dr. Elwyn said he always prefers catgut.

Dr. Bedell said that he has seen several cases and feels that failure to heal has nothing to do with the suture material used. Dr. Kravitz said that he has never been satisfied with any of the explanations offered and felt that it had nothing to do with the sutures. He said that there is something fundamentally wrong in the case which fails to heal. He said that he uses silk sutures at all times.

17. *In the treatment of epithelioma of the lid in older people, is surgical removal preferred to X-ray therapy?*

Dr. Bedell said that the decision must be based on the appearance of the tumor, its apparent thickness and size, and whether its margin is elevated and smooth or ulcerated.

When small, circumscribed, and evidently recent, a correct, single dose of X rays will often cure the disease.

When large, with destruction, a broad dissection is indicated. However, even some of these cases in the very old should be treated by X rays.

Dr. Elwyn said that, whenever possible, he operates, otherwise he uses X-ray therapy. Dr. McLean agreed, and added that, basically, surgical removal is to be preferred except in the very old patient. Dr. Kornzweig felt that surgery should be given more attention; he reported a case in which the patient received X-ray therapy, but then ended up with exenteration of the globe.

18. *Are there any special problems associated with refraction in the aged? Have telescopic lenses or other visual aids been helpful in patients with diminished central vision?*

DR. KRAVITZ said that there are no special problems in refraction of the aged that should give one any trouble. On the contrary, they usually are satisfied with less and so are more easily pleased. However, the few who either have to work or cannot exist without reading can constitute quite a psychic problem if, due to pathologic changes, they cannot be suitably refracted. Usually, patience in explaining the situation to them will bring a favorable response. One must use tact and patience.

Dr. Bedell said it must be explained to patients with senile macular degeneration that they will not go blind; that will satisfy them.

19. *Is there any contraindication to the use of vitamins in the aged? Should they be given generally to old people, regardless of the medical condition?*

DR. MCLEAN said that there is no contraindication to use of vitamins in reasonable doses. Maybe Dr. Zeman should answer the second part of this question.

Dr. Elwyn said that many old patients have clinical and subclinical deficiencies of vitamins; these should be replaced by addition of vitamins. Dr. Zeman said that there is no contraindication to use of vitamins, except possibly their cost. Dr. Kornzweig suggested the use of vitamin B_1 in old patients.

20. *In a program of preventive medicine with reference to the eye, have you any suggestions regarding diet? Use of the eyes? General activity or occupation?*

DR. ELWYN said he could not answer this question. Dr. Kornzweig said that this was part of the general medical problem.

Bernard Kronenberg,
Recording Secretary.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION OF OPHTHALMOLOGY

April 16, 1953

DR. GEORGE F. J. KELLY, *Chairman*

ACUTE HYPOTONY

DR. P. ROBB MCDONALD presented a patient who had had an Elliot trephining operation in the right eye in 1943 for acute glaucoma. The eye was always hypotensive thereafter, but she enjoyed 6/9+ vision until September, 1951, when she was struck in the right eye by an elbow. The vision failed rapidly. There was no evidence of a ruptured cystoid scar, the chamber was formed.

Ophthalmoscopic examination revealed a papilledema and two to three diopters of elevation of the macular region. The vision was hand movements and the tension was too low to record.

Operation consisted of resection of the cystoid scar and closure of the trephination bleb. Recovery of vision was fairly prompt. There was a return of the patient's glaucoma, but this has been controlled by miotics. Gonioscopy revealed a narrow but open angle. Examination of the left eye is essentially negative. Visual fields did not reveal any changes suggestive of glaucoma.

Discussion. DR. EDMUND B. SPAETH: This is a remarkable counterpart of the case that I presented before this section about 10 years ago, though the result in that case was not so fortunate as was this of Dr. McDonald. My patient, the mother of a physician, had had a Lagrange sclerectomy. About eight months later she developed a hypotony so profound that the weight of the lid, plus the pull of the superior rectus muscle caused a deformity of the cornea and of the globe. The sclerectomy incision was closed somewhat similarly to the method used by Dr. McDonald. Very shortly after that the patient developed a marked return of a chronic inflammatory hypertension.

Subsequent surgery was an iris-inclusion operation. The re-established glaucoma was controlled for a short time, but the eye was ultimately lost from the chronic inflammatory secondary glaucoma.

I have not the slightest idea what is the correct therapy for acute hypotony! Severe hypotony has been reported before following corneoscleral trephination. It has been seen after cyclodialysis and after cyclodiathermy. Personally, I know of no such instance developing after an iris-inclusion operation.

It is quite logical to wonder whether this hypotony could be a manifestation of inflammation rather than the result of excessive filtration. If it is the former, then certainly further surgery is contraindicated. If it is the latter, that is, excessive filtration, then one chances a strong likelihood of the re-establishment of the original glaucoma, plus further tissue trauma from further surgery for the hypotony.

The effect which these extreme fluctuations in intraocular pressure have upon the intraocular capillary bed must be serious.

In Dr. McDonald's case there has been a most fortunate outcome. I am very curious to know what the result is going to be in this case after another year. Postoperative hypotony continues inexplicable as to exact cause, controversial as to effect, and unanswered as to treatment.

Follow-up study of glaucoma treated with cyclodiathermy

Dr. Bernard E. Lachman and Dr. Paul A. Rockwell (by invitation) discussed the results of perforating cyclodiathermy in 39 glaucomatous eyes treated according to the method recommended by Castroviejo. All cases were observed between one and two years. Of these 39 cases, 17 had cyclodiathermy as a primary procedure, while, in the remaining 22, cyclodiathermy followed other glaucoma surgery which had failed.

A case was considered successful when:

1. The tension was consistently below 30 mm. Hg (Schiøtz) during the period of observation.
2. When there was no loss of visual field.
3. In case of a blind eye, when there was relief from pain but no atrophy of the globe.

Only seven cases were considered successful by these criteria. No conclusion could be drawn as to the merit of the procedure in any type of glaucoma. All cases required miotics postoperatively, and developed a rise in tension when miotics were discontinued.

Twelve cases of chronic simple glaucoma were treated, eight with failure, four with good results.

Seven cases of glaucoma with aphakia were failures. Two of these subsequently developed atrophia bulbi.

All of nine cases of absolute glaucoma in this series were failures. Three of these developed hypotony and subsequent atrophy of the globe.

Of five cases of congenital glaucoma, all of which had previous glaucoma surgery with failure, two were successfully treated with cyclodiathermy and three were failures.

Five cases were controlled for periods varying between four and 11 months and then developed a rise in tension despite miotics.

Two cases suffered a loss in visual acuity shortly following surgery.

One case of acute glaucoma developed atrophy of the globe 11 months after surgery.

Six cases showed a postoperative rise in tension above pre-operative levels, which persisted despite medical therapy.

Of the 39 cases, six developed atrophia bulbi.

It was concluded that the results of perforating cyclodiathermy by the method which was employed are unpredictable. It is a simple procedure, but not an entirely safe one. It may be followed by a loss of visual acuity, by a persistent rise in tension, and by atrophia bulbi.

Discussion. Dr. Harold G. Scheie:

Cyclodiathermy should be evaluated from two points of view:

1. Results obtained by the original technique; namely, that advocated by Vogt.

2. The effectiveness of the newer methods of the past two or three years.

As you all know, Vogt originally suggested that the electrode should be placed up to within two and one-half to three mm. of the limbus. The present-day techniques suggested by Castroviejo in this country, Weekers and Rieser in Europe, and others, all recommend applying the current six mm. or more from the limbus. In our experience at the Hospital of the University of Pennsylvania, the older method is dangerous because corneal opacity frequently results from damage by the current. The newer technique has proved to be quite safe.

From approximately 1947 until mid-1949, we used cyclodiathermy on 17 patients. The operation failed to control the tension in 10 eyes and corneal opacity due to edema occurred in six. We therefore discontinued use of the procedure until January, 1951. We then adopted the newer technique (the electrode was applied no closer than six mm. from the limbus, nine to 12 applications being made) and used it on 29 eyes.

All of these have been followed for one to two years. The tension was controlled in 14 eyes but miotics were required in four. In several of these patients reoperation was necessary because of a late recurrence of elevated tension. We do not believe the operation is the ideal approach to glaucoma largely because of this tendency. There were, however, no serious complications, as with the original technique. Severe iridocyclitis occurred in one instance with recovery. We have had none of the serious complications mentioned by the author. However, we still reserve the procedure for use when other operations have failed, or may be contraindicated.

Retinopathy of prematurity

Dr. Robert H. Bedrossian (by invitation): The term retinopathy of prematurity, as suggested by Heath, should be used instead of retrolental fibroplasia, since it limits the disease to that condition found in the premature infant, and since most cases of retinopathy do not develop a retrolental membrane.

The classification of the stages of the disease according to Bousquet is thought to follow the clinical course most closely.

The recent literature claiming a relationship between the routine administration of oxygen and the development of the disease is briefly discussed.

Analysis of 44 cases of retinopathy in 127 premature infants under four lbs. showed that the disease developed in the great majority shortly after oxygen supplements were completely withdrawn from the infants.

Thirteen rapidly progressive cases of retinopathy improved within two weeks when the infants were given oxygen therapy. Eleven of the 13 had a recurrence of the disease when the oxygen therapy was stopped.

Statistical analysis showed that the group with retinopathy had oxygen for a longer time but had a shorter weaning from oxygen than the group without retinopathy.

Retinopathy of prematurity appeared in two infants during the course of pneumonia and subsided when the pneumonia resolved.

It was concluded that:

1. Oxygen is not the direct cause of the disease but high oxygen concentrations over a long period of time may predispose to the disease.

2. The most common precipitating cause of the disease is probably the too rapid lowering of the oxygen concentration of the infants' environment.

3. Retinopathy of prematurity may also be precipitated by pneumonias and other respiratory infections.

4. The active vascular phases of the disease may be arrested by placing the infants so affected in the same oxygen environment from which they were withdrawn prior to

the development of the disease.

Discussion. WILLIAM O. LAMOTTE, JR.: Dr. Bedrossian's fine presentation serves admirably to help dispel the doubts of those who still disbelieve in the possible relationship between oxygen and the retinopathy of prematurity. It also points up dramatically the necessity for ophthalmoscopically guided oxygen control of premature infants. I can only corroborate from my own experience the dramatic reduction in incidence of this blinding disease, since the adoption of such measures of oxygen control.

Specifically, as previously reported, the incidence of this disease in bilateral blinding form at the Delaware Hospital in Wilmington, during the four-year period of 1948 through 1951, was 33.8 percent in all infants four pounds or under at birth. This incidence did not vary with various forms of therapy, including vitamin E and ACTH, the incidence for example in 1951 being 38.8 percent.

Beginning January 1, 1952, we adopted the suggestions and methods of Szewcyck, with a striking reduction in the incidence of the disease during the year 1952, to 3.7 percent. This was based on 27 infants under four pounds at birth, 18 of whom showed various grades of the retinopathy of prematurity.

This is consistent with the expectation that two thirds of all infants of this birth weight group will show some signs of this disease.

Normally, it would be expected that half of these would return to normal spontaneously, and half would show progression to permanent changes. Actually, only four of the total show any permanent changes at all, and three of these will have normal vision in at least one eye, while only one of the whole group has bilateral retinal detachments; it is the one infant which gives us the incidence of 3.7 percent.

One cannot help but be impressed by the fact that the incidence of this disease in blinding form had dropped from one out of three to one out of 27, since the adoption of these methods.

I agree with Dr. Bedrossian's conception of a secondary anoxia being the chief mechanism involved in this disease. I would take exception, however, to his implication that these eyes are usually normal while the infants are still in oxygen.

Most of these eyes are abnormal because the retinal arterioles, although not engorged and tortuous, are likewise not normal because they are usually quite narrow. As reported to this group in December, 1949, extreme narrowing and attenuation of the retinal arterioles is one of the first signs of this disease.

Although most all infants who have been in high oxygen for any length of time will show narrowing of the arterioles, those who show the most extreme attenuation may assuredly be suspected of being those most likely to develop the retinopathy of prematurity after removal from oxygen. This is therefore an advance notice of those infants who are probably the most unstable and most sensitive to metabolic changes.

If anyone should doubt the relationship of oxygen to the caliber of the retinal vessels, let him take, for example, an infant weighing five and one-half pounds at birth, in whom the chances of precipitating permanent change are remote, and keep the baby at 90-percent oxygen for several days, and then suddenly remove him to room air. He will see, within the course of one to two days, an increase in the caliber of the retinal vessels to two or three times normal size, which, on re-introduction into high oxygen concentration, will return to normal in another two or three days, a process requiring a week to 10 days without the reintroduction into oxygen.

I would like to conclude with the following axioms which seem to be applicable to this situation. Premature infants in the weight group predisposed to the development of this disease should receive oxygen in the lowest concentration for the shortest

period of time compatible with their survival. Except in the most unusual circumstances, the oxygen concentration should never be above 50 percent.

Oxygen concentration should be determined by actual measurements of the oxygen concentration and not upon liter flow. For example, the same liter flow per minute in two different isolettes has been found to vary as much as 30 percent.

The value of an Isolette is not in the high concentrations of oxygen which can be developed but in the fact that stable and steady oxygen concentrations can be maintained without wide fluctuations, which tend to activate the instability of these primitive vascular systems.

Careful ophthalmoscopic control has become a necessity in the management and conduct of a premature nursery.

Chronic progressive external ophthalmoplegia

Dr. Gabriel A. Schwarz and Chan-Nao Liu, Ph.D. (by invitation): There is a disease of man characterized by weakness of the muscles of the eyelids and weakness of the external muscles of the eyeballs. This disease is slowly progressive over many years. There is occasional familial incidence. Some authorities have felt that the condition was the result of disease of the neurones in the brain stem which supply these muscles. Others, more recently, have emphasized the resemblance of this condition to muscular dystrophy.

Our patient, a white man, died at the age of 63 years from a spongioblastoma polare of the left parietal lobe. At the age of 27 years, he had begun to have drooping of his left upper eyelid; at 41 years, ptosis had begun on the right side. The onset of weakness of the movements of his eyeballs had come so insidiously that no definite time of onset could be established. When examined a year before his death, the patient showed bilateral ptosis, eyeballs in primary position, only barely perceptible movements of his eyeballs either voluntarily or reflexly, pupils which were equal and regular and round and reacted well to light but poorly to attempted convergence. The other cranial nerves were intact.

Neuropathologic studies of the oculomotor, trochlear, and abducens nuclei showed (1) the cellular populations to be unchanged, (2) the motor neurones to be somewhat smaller, less polygonal and rounder than usual, (3) cytoplasmic clumps of granular yellow-brown pigment, and (4) vacuoles in some perikaryons. In the abducens nuclei, chromatolysis was also noted but there were many more intact neurones. One of the masticator nuclei contained neurones with definite chromatolysis.

The intra-axial portions of the oculomotor, trochlear, and abducens nerves were readily identified in myelin preparations and showed no demyelinization. The extra-axial portions of these nerves also showed no demyelinization. The oculomotor nerves seemed to have a general overabundance of nuclei throughout. The axis cylinders of the right trochlear nerve were counted and compared to the number in a normal control; the difference in the fiber counts was not significant.

The left superior oblique, the left superior rectus, and the right lateral rectus muscles were found to show a marked and diffuse destruction and loss of muscle fibers. The remaining muscle fibers showed many and varied changes. Large and small fibers were seen. Myofibrils in some were swollen and torn apart. Portions of muscles were hyalinized. Areas of muscles were granular. Some few fibers looked fairly normal. Even in one muscle fiber, there were these varied alterations.

There was a marked increase in the sarcolemmal nuclei about the muscle fibers. Fat and connective-tissue replacement and overgrowth occupied much of the muscles. The myelin stain and the axis-cylinder stain disclosed abundant intact intramuscular nerve fibers.

Motor end-plates could be identified on severely degenerated muscle fibers or apparently free in the loose connective tissue. Elaborate spiral nerve endings were also found about degenerating muscle fibers.

Both ciliary ganglia were studied. Finely myelinated nerve fibers were found. The ganglion cells showed the usual histologic variations seen in these autonomic neurones, but no marked or consistent pathologic change.

The establishment of any particular wasting muscular disease as being "primary" or "secondary" is not always simple. More recent studies have emphasized the need to observe the intramuscular nerve fibers and nerve endings. Such studies had never been done before in this disease.

In our case, the changes in the extra-ocular muscles were typical of changes described late in muscular dystrophy but the significant finding was the preservation of abundant nerve fibers and the presence of motor-nerve endings in association with an intense muscle fiber destruction.

These findings, coupled with the preservation of the intra-axial and extra-axial nerve fibers supplying the disease muscles and the relatively healthy motor nerve cell bodies of the nuclei, which gave rise to the motor nerves to the abnormal muscles—all of these findings considered together seem to us to favor the opinion expressed by Kiloh and Nevin (1951) that chronic progressive external ophthalmoplegia is a form of muscular dystrophy and could be called rightfully "ocular myopathy."

Discussion. Dr. Francis Heed Adler. The question at issue is whether the pathogenesis of chronic progressive ophthalmoplegia is primarily a lesion in the nuclei of origin of the ocular muscles or is in the muscles themselves. Wilbrant and Saenger, who first collected a series of these patients, suggested that the disease was primarily due to a degeneration of the cells in the nuclei of the third, fourth, and sixth nerves.

This was borne out by the pathologic report of a case by Dr. Langdon and Dr. Cadwalder. These authors found a diminution in the number of cells in the nuclei of the sixth nerve in their patients, but were uncertain about the counts in the third nuclei. They did not examine the ocular muscles themselves.

It is obvious that, in order to decide this question, one must make an examination of the nuclei of origin of the nerves, the nerves themselves, and the muscles. Dr. Schwarz had done this and has found no changes in the nuclei as far as cell count of degenerative changes in the cells. The nerve fibers themselves were not reduced in number. However, he did find changes characteristic of myotonic dystrophy in the muscles themselves.

Unless we are dealing with two separate disease entities with different pathogenesis, one must assume that the cases of so-called chronic progressive ophthalmoplegia belong in the group of ocular myopathies, and are due to some primary disturbance in the ocular muscles themselves.

M. Luther Kauffman,
Clerk.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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XVII INTERNATIONAL CONGRESS

The final touches on the program of the XVII International Congress of Ophthalmology have been made, and the picture stands out as the most complete coverage of ophthalmology that has ever been attempted. Extensive technical and scientific exhibits from home and abroad are spread over the rooms adjacent to the Grand Ballroom and the room where motion pictures will be exhibited every afternoon, Monday through Friday. Televised eye clinics from the New York Eye and Ear Infirmary will be shown in the Grand Ballroom every afternoon, Tuesday through Friday. At scientific sessions, morning and afternoon, approximately 200 papers will be presented and discussed, mostly clinical, of interest to every practitioner of ophthalmology in the world.

All papers are abstracted in English, French, and Spanish. More than 80 percent of them will be read in English. More than

500 oculists from abroad have registered in advance, and all continents are represented. Three thousand American ophthalmologists are expected to register for this, the most comprehensive congress of ophthalmology ever held, all under one roof. Hotel space is not a problem.

William L. Benedict.

THE JUNE MEETINGS

Ophthalmologists attending the 1954 meetings of the American Medical Association, the Association for Research in Ophthalmology, and the American Ophthalmological Society were rewarded with a wide variety of interesting scientific papers, frequent vigorous discussions, and many pleasant exchanges of clinical experience. At the conclusion of two weeks of travel, much western hospitality, and many meetings, almost everyone was prepared for a vacation or a rest at home. Some 60 papers were presented at the three meetings and they represented the best of ophthalmic study and experimentation during the past year.

The 90th annual meeting of the American Ophthalmological Society was held at Glacier National Park, June 16th, 17th, and 18th, under the presidency of William L. Benedict. The members and guests were rewarded for their long journey with unforgettable views of snow-capped peaks, mountain lakes, and the rugged grandeur of the Rockies. The following day, they awakened to a snow storm, which continued intermittently the remainder of the meeting. Indoor activities before the giant fireplaces proved more attractive to most than going outside, as some saw the worst winter in years.

The program was unusually interesting, with the papers stimulating a free and occasionally vigorous exchange of ideas.

The Howe Medal was awarded to John H. Dunnington, director of the Institute of Ophthalmology of Presbyterian Hospital and Columbia University, for his proficiency as a physician, teacher, researcher, and administrator. The affection and respect in which he is held was mirrored in the applause which followed the announcement of the award.

Everett L. Goar was elected president and Alan C. Woods vice-president. Maynard C. Wheeler and Gordon Bruce were re-elected secretary and editor, respectively. The next meeting will be held at the Greenbrier at White Sulfur Springs June 2, 3, and 4, 1955.

The scientific assembly of the American Medical Association met in San Francisco June 21st to 25th and the successful joint meeting of the Section on Ophthalmology and the Association for Research was continued. This union of basic and applied studies yields more interesting meetings annually and an unusually large number attended. The microphone was used for all discussions and replies and there was a complete absence of colloquies between the speaker and those near by, which was a delightful and long-awaited innovation.

The weather in San Francisco was pleasant and the scenic and dining attractions numerous, so that every minute was filled. Approximately 220 scientific and 300 commercial exhibits competed for attention. William F. Hughes and his committee selected eight exhibits in ophthalmology and many studies in other sections were of ophthalmic interest. A certificate of merit was awarded to Aleta N. Barber, G. N. Ronstrom, and R. J. Muelling, Jr., of Louisiana State University for their exhibit of "Development of the visual pathway in humans." Honorable mention went to David O. Harrington and Milton Flocks of the University of California and Fort Miley Veterans Administration Hospital for "The multiple-pattern method of visual-field examination."

Dr. Trygve Gundersen, in the chairman's address, pleaded for the prevention of visual loss by routine tonometry in all patients past 40 years of age and by routine visual acuity measurement in children, beginning at the age of three years. Dr. Arthur J. Be-

dell was introduced as guest of honor of the Section, and then spoke on choriodopathy in his customary vigorous, well-illustrated, and incisive manner.

The Medal for Distinguished Service to Ophthalmology was awarded to Otto Barkan for his many contributions for ophthalmology, particularly in the classification of the glaucomas and in the popularization of goniotomy for congenital glaucoma.

Erling W. Hansen and Watson Gailey were elected chairman and vice-chairman, respectively, of the Section of Ophthalmology of the American Medical Association. Dr. Harold G. Scheie was re-elected secretary. The next meeting of the American Medical Association will be in Atlantic City in June, 1955.

At the annual banquet of the Association for Research in Ophthalmology David G. Cogan, director of the Howe Laboratories for Ophthalmic Research of Harvard Medical School, was awarded the Proctor Medal for his continued and long time excellence in ophthalmic research. In a gracious acceptance, Dr. Cogan described the minimal training required for effective ophthalmic research. Brittain F. Payne and James H. Allen were honored for their services to ophthalmology during their terms as secretary of the Association for Research. At the annual business meeting, James H. Allen was elected trustee and was succeeded as secretary by Lorand V. Johnson.

Several subjects appeared to be of widespread interest, judging by the number of papers and the discussions which they stimulated. Radioactive phosphorus may prove to be valuable in the differentiation of ocular tumors but at present studies are beset with technical problems. Herpetic keratitis and the sterilization of instruments and ophthalmic medications are the subjects of research at a number of institutes. Diamox is being intensively studied by a variety of methods and seems likely to be as useful as a research tool as a therapeutic agent.

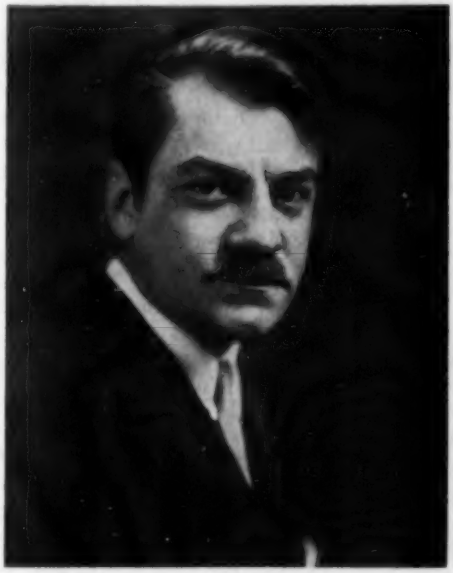
Most of those present agreed that the San Francisco meetings were most successful and, despite criticism of the hotel arrangements, all seemed anxious to return to San Francisco for another meeting in 1958.

Frank W. Newell.

OBITUARY

LeGRAND H. HARDY
1895-1954

LeGrand H. Hardy died on April 14, 1954, at the age of 59 years, after a long heart illness. He was clinical professor of ophthalmology in the College of Physicians and Surgeons of Columbia University and associate attending ophthalmologist of Presbyterian Hospital and director of the Knapp Memorial Physiological Optics Laboratories at the time of his death. Previous hospital connections included the New York Eye and Ear Infirmary, the Northern Dispensary, the Mid-Town Hospital, the Fifth Avenue Hospital as director of ophthalmology, and, finally, the Institute of Ophthalmology and Vanderbilt Clinic of Presbyterian Hospital.



LeGrand H. Hardy, M.D.

He was a member of the New York Academy of Medicine, the New York Ophthalmological Society, the American Ophthalmological Society, the Association for Research in Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, a diplomate of the American Board of Ophthalmology, and a fellow of the American College of Surgeons.

His written contributions to ophthalmology were not numerous but they were respected for their quality. His main interests lay in the field of physiologic optics in which he was recognized as an authority. During the past few years, he was engaged in a study of the geometry of binocular space perception for the Office of Naval Research. During World War II he served with the Office of Scientific Research and Development of the Armed Forces. His most recent contribution, finished but as yet unpublished, deals with color anomalies and their detection. A pioneer in orthoptic training, he was president of the Orthoptic Council, which he founded in 1938, for 10 years.

LeGrand Hardy was born in Provo City, Utah, of illustrious parents. His father was one of the first trained medical men in the state of Utah where he was head of the state mental hospital for many years. His mother was a sister of Senator Reed Smoot. His maternal grandfather was responsible for the organizing and financial backing of what is now known as the Brigham Young University.

LeGrand Hardy was graduated from Brigham Young University in 1916. After graduate study at the University of Chicago, he received a Bachelor of Science degree from Columbia University in 1919 and a medical degree in 1921. Post-graduate study in Germany and Switzerland followed.

He always did well in school and, even at an early age, a great intellectual future was predicted for him. Like many able people he did not confine himself to scholastic achievements but took a large part in extracurricular activities.

It was only natural that he should have a tradition of high moral impulses combined with a fearlessness in the intellectual as well as in the physical world. He was never interested in the trivia of personal behavior, but simply expected honesty in others as he himself believed it and practiced it. It was the world about him that was exciting.

Always interested in the out-of-doors and any challenge that it presented, he was a member of the first small party to climb Mt. Moran of the Teton Range in Wyoming. With other boyhood friends he was always climbing the mountains in Utah which he loved. At other times he worked as a cowpuncher, or in the mines, where he became expert in the handling of dynamite.

Among the first organizers of the Boy Scout movement in Utah it was characteristic of him that he did not feel qualified as a leader until he could surpass all of the scouts under him in attaining merit badges.

His zest for living and his insatiable intellectual curiosity never left him. His interests were universal, including history, religion, and the arts, as well as pure science. Careful and painstaking in his own work he always respected and learned from even the lowliest craftsmen who did a good job. It was difficult to find a topic of conversation with which he was not conversant and unusually well informed.

Intolerant of sham or ostentation in any form, LeGrand Hardy was uncompromisingly honest with himself and with others. He knew his own shortcomings and made no apologies for them unless, in any way, they caused distress to others. To anyone who needed his help he was counselor and friend as few men could be. His loyalty was intense.

In 1923 he was married to Susanna Haigh who survives him.

Ophthalmology has lost a brilliant mind and a careful worker but those who were privileged to call him friend have suffered a greater loss.

Willis S. Knighton.

CORRESPONDENCE

Astatine dosage

Editor,
American Journal of Ophthalmology:

In my article on "Alpha irradiation of the anterior segment" which was printed in The Journal, **37**:183 (Feb.) 1954, the dosage of astatine used appears to be presented in millicurie amounts, when it should be in microcuries.

It is estimated from animal studies that the lethal dose in man may be as low as 50 millicuries of astatine, whereas 50 microcuries would probably be quite safe. I would appreciate it if you would print this correction in the hope of preventing any experimenter from using too high dosage in the future.

(Signed) Robert N. Shaffer,
San Francisco, California.

Bücklers: A reappraisal

Editor,
American Journal of Ophthalmology:

In a letter just received from Prof. Jean Nordmann of Strasbourg he writes that at the recent meeting of the Congrés de la Société Française d'Ophtalmologie he met Prof. A. Franceschetti of Geneva and in a personal discussion mentioned my editorial on German Ophthalmology (The Journal, **37**:124, 1954). Both agreed wholeheartedly with its content except for the implication that Bücklers' classic work on hereditary corneal dystrophies was motivated by Nazi ideology. This inference had been expressed in the American articles that discussed Bücklers' contribution (The Journal, Goar, E. L.: **33**:674, 1950; Clark, W. B.: **33**:692, 1950).

According to Franceschetti, this inference is not justified. He has known Bücklers very well and can vouch for his exemplary behavior during and after the war. Of the German oculists, Bücklers was indeed one of the most anti-Nazi and did not get a professorship because of his anti-Hitlerism. Now, because of his famed article on heredity, he is considered an old-time Nazi and cannot obtain a university connection.

(Signed) James E. Lebensohn,
Chicago, Illinois.

BOOK REVIEWS

Parsons' Diseases of the Eye. By Sir Stewart Duke-Elder, K.C.V.O., etc. New York, The Macmillan Company, 1954, edition 12. 606 pages, 22 color plates, 465 text figures, index. Price: $8.00.

The many recent advances in ocular physiology, the etiology and pathology of ocular disease, the ocular manifestations of general disease, and particularly in the field of ophthalmic therapeutics, have required of the author the task of thoroughly revising, rearranging, and, in many places, of completely rewriting this well-known and justly popular textbook of diseases of the eye. Many of the old illustrations have been replaced by better ones, and many new and excellent ones have been added. At the same time, mirabilis dictu, the total size of the book has been considerably reduced. This is a most notable achievement.

Many students of ophthalmology in the United States have not been as familiar with earlier editions of this fine textbook as have English-speaking students elsewhere in the world, particularly in Great Britain. I believe the reason for this has been its competition here with other textbooks of an introductory nature which are smaller, austere, more compact, and essentially designed for the student of medicine who has to read while he runs, and to whom the relatively few hours assigned to the teaching of ophthalmology seem to him to be an index of its importance in relation to other branches of the medical studies. This has been a pity, for the previous editions, written by a world-famous scholar, Sir John Parsons, contained much information of value to the student,

and particularly to the general practitioner, that the smaller textbooks were, of necessity, unable to supply. One could argue further and say that perhaps this is one of the reasons why general practitioners in this country at least seem to know so little about the eye and, in some instances, are even indifferent to ophthalmology.

Sir John, beloved by all, who still comes out of his retirement near Leeds on too rare occasions, and delights his many devoted friends, admirers, and colleagues with apt discussions of ophthalmic topics and brilliant exercisings of his acute and rare intelligence, has turned over the burden of producing this textbook to Sir Stewart. A more suitable and worthy heir could not have been chosen. The sublimely majestic genius of Sir Stewart in collecting, sifting, editing, and collating facts and most skillfully discarding the fancies of our science is too well known for further comment. His beautifully lucid and flowing style of writing that so delights us in his supercollosal *Textbook of Ophthalmology*, and indeed in any article or book to which he turns his hand, continues to give us pleasure and profit in this present task of his which somehow or other, and no one knows how, he has found time to do most accurately.

While primarily designed "as a concise and up-to-date introduction to the diseases of the eye for students, general practitioners, and junior ophthalmic surgeons," this fine book should be the vade mecum and bedside companion of every ophthalmologist.

Derrick Vail.

OPHTHALMIC PLASTIC SURGERY. By Sidney A. Fox, M.D., New York, Grune and Stratton, Inc., 1952. 290 pages, 133 figures, bibliography, and index. Price: $15.00.

This new text is not presented as an encyclopedia on plastic surgery but rather as a compilation of techniques and procedures which the author considers to be especially valuable and which have proven to be successful in his hands.

The first two chapters describe surface and surgical anatomy of the brow and lids and the fundamentals of lid surgery such as lid splitting, halving, plasties, excision of scars, and skin closures. The section devoted to grafts not only includes the different types but valuable directions are included to aid the surgeon in his preparation and placement of the grafts. It is gratifying to note that the author prefers to use free skin grafts whenever it is possible to use them. The proper application of pressure and pressure dressings is stressed.

Minor surgical procedures, first aid to the lid margin, and the repair of notches and colobomas have been given considerable attention. Numerous drawings are presented to show the methods for creating temporary and permanent tarsorrhaphies and the principles of Wheeler's halving procedure are stressed and applied in the repair of colobomas and associated defects.

Entropion and ectropion are discussed adequately. The author prefers Wheeler's orbicularis transplant in the repair of spastic entropion although he does devote some space to a procedure in which tarsus, skin, and muscle are excised. The latter embodies the principle of the tarsal triangle described by Butler but additional skin and orbicularis are resected to increase the effect. Several procedures are recommended for senile ectropion, the common Kuhnt-Szymanowski being given first place. Cicatricial ectropion he repairs by sliding grafts or by the replacement of lost tissues with rotated pedicle or free whole skin grafts.

Many of the common ptosis procedures are discussed and he agrees with others who favor strengthening the lid levator wherever possible.

The rest of the book is devoted to chapters on displacement of the lid and globe, the conjunctiva, socket, and upper and lower lid reconstruction. Few will disagree with the

statement that the Hughes technique is one of the best means at our disposal for partial or total lid reconstruction.

The book is well written and it will appeal to the practicing ophthalmologist who is interested in plastic surgery and in operations that can be used in everyday practice. The drawings emphasize clarity and simplicity rather than fine art work.

Irving Puntenney.

THE ANTISEPTIC, April, 1954.

The golden jubilee number of *The Antiseptic*, its April, 1954, issue, is an impressive volume of over 700 pages which contains a series of summaries of the achievements of the last 50 years in each of the special fields of medical interest, among them ophthalmology, and also many original essays by contributors from many lands. There are four ophthalmic articles on immunologic therapy, on vitamin-A deficiency, on retinal detachment, and on the plastic lens in cataract surgery.

F. H. Haessler.

TUBERCULOSES OCULAIRES ET TUBERCULOSES PARAGANGLIONNAIRES. By L. Paufique and J. Brun. Paris, Masson et Cie., 1953. 186 pages, 43 illustrations. Price: 1,350 francs.

This monograph is the product of the close collaboration of Paufique, ophthalmologist, and Brun, phthisiologist, both associate professors at the Lydon Medical School. The limitations of clinical and laboratory examinations are thoroughly discussed. Up to the age of six years, a positive von Pirquet test is practically diagnostic. The excellent chapter on differential diagnosis fails, however, to mention toxoplasmosis.

In the 15 pages devoted to therapy the authors stress that streptomycin is still the most important chemotherapeutic agent. Tuberculin is administered on a tentative basis and abandoned if no amelioration is evident after a dozen injections. But if favorable results follow, they advise three series of 16 injections the first year, two such series the second year, and then one series annually for the next three years. Large doses of vitamin D are considered valuable adjuvant treatment in tuberculous iritis of middle age and in tuberculous periphlebitis.

The term "paraganglionnaires" is a neologism, introduced to emphasize the interdependence of certain types of ocular tuberculosis with tuberculous adenopathy, and this concept is elaborated in 82 pages. The tuberculous adenopathy is neutralized by X-ray treatments, which indirectly act on the tubercle bacilli by the tissue reaction provoked. For peripheral adenopathy, as in the cervical glands, 150 kv. is advised with a filter of 0.5-mm. copper; for mediastinal adenopathy the same filtration is used with 180 kv. A series of five treatments of 25 r to 100 r is given at weekly intervals and after a month the series is repeated. The tuberculin reaction in the skin does not necessarily reflect the allergy of the eye. The tubercle bacillus undergoes profound changes in the lymph glands which in turn become the pivot of the phenomena of allergy and immunity.

James E. Lebensohn.

ARCHIVES OF THE OPHTHALMOLOGICAL SOCIETY OF NORTHERN GREECE. Thessaloniki, Greece, 1952.

This is the first volume of the newly formed Ophthalmological Society in Northern Greece, and it is a welcome contribution to the Greek ophthalmic literature. It contains 26 reports and presentations of cases.

Zervakakos reports a case of oculogyric crises in a 35-year-old man and emphasizes its diagnostic significance in Parkinsonism. The patient apparently suffered from a hyperkinetic form of encephalitis three years previously.

Karagounidis recommends the early use of aureomycin in herpetic keratitis. He had a

favorable response in five cases from local and systemic application of aureomycin. However, he noticed no improvement in disciform herpetic keratitis.

Polychronakos reports an interesting case of recurrent unilateral exophthalmos in a four-year-old child due to spontaneous orbital hemorrhage. The laboratory studies and physical examination were negative and no signs of scurvy were present. Full recovery was accomplished without any specific treatment. The author believes rupture of a cirsoid orbital aneurysm is a possible cause.

Georgiades reports in detail the case of an intermittent unilateral exophthalmos in a seven-year-old child due probably to a congenital vascular anomaly of the orbit.

Konstas reviewed the bibliography and reports three cases of cataracta centralis pulverulenta. Pedigree of the family indicates a dominant type of heredity.

Georgiades reports a case of Duane's retraction syndrome in a 19-year-old girl and a case of oxycephaly associated with secondary optic atrophy.

Raptis treated successfully a case of postherpetic disciform keratitis with local and subconjunctival injection of cortisone.

Konstas presents a posttraumatic ischemia of the retina simulating occlusion of the central retinal artery in a young girl. He thinks that hematoma into the sheath of the optic nerve was the cause of the retinal ischemia due to compression of the central retinal artery.

Raptis was able to arrest the progression of two cases of Mouren's corneal ulcer with cautery; he also treated a case of Eales' disease with irradiation of the spleen which showed no recurrence for a year.

Polychronakos reports a case of recurrent orbital edema probably of allergic etiology in an enucleated eye.

Konstas presents four cases of Duane's retraction syndrome and also an interesting case of oxycephaly associated with facial hemiatrophy, bilateral ptosis, bilateral sixth nerve paresis, coloboma of the choroid, and persistent hyaloid artery.

Polychronakos discusses in detail the free grafting in correcting cicatricial ectropion and presents 10 cases successfully treated by this method, employing Padgett's dermatome.

Georgiades reports a rare case of subretinal cysticercus which was followed by him for 16 months. The first symptom was sudden loss of vision due to a retrobulbar neuritis, followed by discoid retinal hemorrhage. A month later the parasite was visible in the fundus. No attempt was made to remove the parasite and the eye was lost. The same author presents a case of primary epithelioma of the orbit completely removed and postirradiated with no recurrence for three years.

Konstas recommends tarsectomy, partial or total, in cases of trichiasis, but also as a preventive operation in the serious complications of trachoma III and IV. Only in the latter stages is tarsectomy indicated.

Polychronakos reports a case of unilateral exophthalmos associated with ptosis due to a cirsoid aneurysm of the superior ophthalmic vein. Excision after ligation of the vein decreased the exophthalmos. The same author presents a case of primary tuberculosis of the tarsal conjunctiva of the upper lid which subsided completely three months later after the application of streptomycin systemically and Rimifon and PAS ointment locally.

Georgiades, Petridis, and Kalaitjis also report another rare case of secondary tuberculosis of the tarsal and bulbar conjunctiva consisting of a small yellowish nodule. The patient had a recurrent tuberculous kerato-iritis for five years of the same eye.

A brief summary in French follows at the end of the book.

Manos A. Petrohelos.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

7

CONJUNCTIVA, CORNEA, SCLERA

Chams. **Electrocoagulation of trachoma.** Rev. intern. du trachome **31**:1-46, 1954.

Chams and his collaborators have treated more than 200,000 cases of trachoma since 1930 by electrocoagulation of the conjunctiva and cornea. The entire conjunctival sac is coagulated by means of a ball electrode of 2.5 mm. diameter and a current of from 200 to 250 milliamperes. The author states that the coagulated conjunctiva does not whiten and that the scar is always smooth. Biomicroscopic, cytobacteriologic, histologic, and inoculation studies before and after coagulation are reported. Chams describes a drop in the trachoma index in the city of Dezfoul from 95 percent in March, 1949, to 37 percent in May, 1953, and in the city of Teheran from 47 percent in 1936 to 1 percent in 1953. (28 color plates, 6 tables, 24 references)

P. Thygeson.

Decour, H., Ferrand, G., and Reinhards, J. **Effect on the evolution of trachoma of mass treatment of the acute ophthalmias by instillation of 1-percent aureomycin.** Rev. intern. du trachome **31**:119-140, 1954.

In 1952 the service for the prophylaxis of ocular disease of the Moroccan Public Health Department carried out a study on the effect of prophylactic treatment of acute conjunctivitis in 9,465 persons. The treatment consisted in two applications daily of 1-percent aureomycin ointment for three days every three weeks. From the total series a group of 2,994 individuals were selected for special investigation, 1,566 of them being subjected to treatment and 1,428 left as controls. Periodic surveys of this group showed that aureomycin was a useful prophylactic and that it also cured bacterial conjunctivitis within three days. The authors found that Koch-Weeks conjunctivitis had a definite tendency to reactivate quiescent trachoma, and that prophylaxis of epidemic conjunctivitis had a favorable effect on trachoma and favored the natural tendency of the disease to heal spontaneously. In conclusion they advocate mass education for the use of prophylactic antibiotic medication in areas subject to epidemic conjunctivitis. (4 tables)

P. Thygeson.

François, J., and Neetens, A. **Recurrent dystrophic erosion of the corneal epithelium.** Ann. d'ocul. **187**:237-254, March, 1954.

Recurrent corneal erosion is a true epithelial dystrophy, the primary cause of which is not known, trauma being only an occasional cause. The spontaneous forms are usually bilateral. Women are more often affected than men, and the condition is usually observed between 25 and 40 years of age. The condition recurs at irregular intervals, having its onset in the morning on awaking. It is always localized below the pupillary area and not necessarily at the site of trauma. Between recurrences, there persists a latent epithelial dystrophy, characterized subjectively by pricking sensations in the mornings and, objectively, by gray epithelial spots below the pupillary area.

Histopathologically, vacuolization of the epithelial cells with pyknotic or absent nuclei is seen in some places, and in others increased basophilia with multiple nuclei and basophilic granulations.

Lamellar corneal transplants have given the authors their best results. They used a 3-mm. transplant. Four cases treated by this method have been cured after the first treatment. Among many other methods of treatment described, only three should be retained: 1. the instillation of 1 to 2-percent silver nitrate after removing the lower half of the corneal epithelium; 2. total removal of the epithelium with a curette or knife after cocaine anesthesia; and 3. galvanocauterization of the erosion, which, by provoking an inflammatory reaction, favors adherence of the epithelium to the cornea proper. This last method, while very effective, has the disadvantage of producing a corneal scar. (5 figures, 65 references)

John C. Locke.

Goar, E. L., and de la Motte, G. W. **Cystine crystals in the cornea and conjunctiva.** A.M.A. Arch. Ophth. **51**:336-343, March, 1954.

Cystinosis is a fatal disorder of protein metabolism occurring in infants and children. It is also known as de Toni-Fanconi syndrome, renal rickets, or intractable rickets. Two cases are reported in which the diagnosis was made by examination of the corneas with the biomicroscope, which revealed innumerable iridescent crystals embedded in the superficial layers of the cornea. The authors advise such an examination in all cases of severe rickets. (5 figures, 8 references) R. W. Danielson.

Gupta, S. P. **Modern treatment of trachoma by chloromycetin.** Rev. intern. du trachome **31**:182-192, 1954.

In a study of 400 cases of trachoma treated by chloromycetin topically in solution and ointment, and systemically by oral capsules, Gupta obtained cures in 100 percent of cases of Stage I trachoma. In chronic trachoma the instillation of 1-percent ointment six times daily gave cures in about 75 percent of cases; treatment time could be shortened by the simultaneous use of follicular expression. Systemic administration of the drug offered no advantages. In cases which relapsed, a second course of treatment proved effective. P. Thygeson.

Hanser, S. Albert. **Use of cortogen and chlor-trimeton maleate in the treatment of allergic conjunctivitis.** Tr. Am. Acad. Ophth. **58**:133-135, Jan.-Feb., 1954.

In simple allergic conjunctivitis, cortogen and chlor-trimeton maleate used locally in the eye as drops caused a greater drop in the eosinophile count on scrape smears from the conjunctiva and gave better subjective improvement than other therapeutic agents used.

Theodore M. Shapira.

Hobbs, H. E. **Three therapeutic grafts.** Brit. J. Ophth. **38**:61-63, Jan., 1954.

For chronic and recurrent corneal disorders lamellar grafting possesses a therapeutic influence the nature of which is not quite understood. Three cases are re-

ported in which the patient received considerable benefit. All three presented long-standing resistant corneal edema, which was painful and which no longer reacted well to cortisone. In each case a 7-mm. lamellar graft healed uneventfully with relief of pain and inflammation. (2 figures, 10 references) Morris Kaplan.

Kozlowski, Bogumil. **Unusual form of hyaline degeneration of the cornea.** Klinika Oczna **23**:249-253, 1953.

A case of hyaline degeneration of both corneas in a woman, 40 years of age, is described. The condition developed gradually, bringing visual acuity down to finger counting at 15 to 20 cm. The corneas were foggy with a granular surface and superficial vascularization. Conservative treatment did not improve the condition of the eyes. Both corneas were curetted and part of the superficial layers were peeled off. The result was gratifying. The vision improved to 4/36 and 4/24 in the right and the left eye respectively. Microscopic examination revealed changes in the epithelium and in the stroma. They were characterized by areas of degenerated confluent uniform masses. (3 figures, 8 references) S. Brandon.

Leigh, A. G. **Opacification in perforating corneal grafts.** Brit. J. Ophth. **38**:10-18, Jan., 1954.

Ideally the cornea to be grafted should possess clear, normal tissue surrounding the opacity; this, however, is not always possible although patches of normal cornea among the opacities brighten the outlook very much. If the cornea is completely opaque then the graft is almost sure to become opaque. Vascularization of the cornea does not interfere seriously if it consists of one or several large isolated vessels; numerous small vessels tend to invade the graft. If the graft is dislocated, if vitreous is lost, if infection or anterior synechia result, then the graft is almost sure to become opaque. In two cases in which the graft remained quite clear and then apparently suddenly became completely opaque, regrafting was completely successful; the opaque grafts were found to have bands of fibrous tissue spread over the posterior surface. The technique recommended involves punching out rather than trephining and the use of overlay sutures rather than direct suturing since direct sutures traumatize the graft. The eyes are dressed within 36 to 48 hours and the corneas are freed of all mucus and debris daily. The instillation of cortisone drops is begun on the 10th day and continued for several weeks. (8 figures, 1 reference) Morris Kaplan.

Majoros, Manos. **Etiology and treatment of trachoma.** Rev. intern. du trachome **31**:173-181, 1954.

The author comments on his conception of the etiology and treatment of trachoma and advances the hypothesis that the disease may be a general disorder because of the greater efficacy of systemic sulfonamide therapy than of topical therapy. He describes his present method of treatment which consists of grattage of the follicles with a dry cotton swab, combined with oral sulfonamide therapy. He believes that all mutilating operations should be abolished. P. Thygeson.

Mandic, Ljubisa. **On the classification of trachoma.** Rev. intern. du trachome **31**: 221-234, 1954.

A new classification of trachoma, differing markedly from the standard MacCallan classification, is proposed by the author. It is based on the location of the major involvement of the disease, Tr. I indicating involvement of the upper tarsal conjunctiva, Tr. II involvement of the conjunctiva of the upper tarsus and fornix, Tr. III involvement of the conjunctiva of both eyelids, of the fornices, and of the upper half of the cornea, and Tr. IV

involvement of the whole conjunctiva and cornea. Cicatricial trachoma would be designated by the letters CIC, followed by Roman numerals indicating the site of major cicatrization. P. Thygeson.

Michaelson, I. C. **Slope of sides of corneal grafts and recipient beds.** Brit. J. Ophth. **38**:19-21, Jan., 1954.

Improper coaptation of a corneal graft to its recipient bed may result in unwelcome complications and it is easily seen that there may be great variation in the slope of the sides of both the graft and the bed. A series of studies was made on cadaver eyes to determine what influences this slope. The eyeball was held in the fingers with and without pressure exerted against it, and the grafts were made from the epithelial as well as from the endothelial surface. Each method produced a different slope to the sides of the corneal button. These slopes were vertical, convergent or obliquely parallel depending on these factors. The slope of the sides of the recipient bed was almost universally a slope inwards, which does not always match the slope of the graft. (2 figures, 1 reference) Morris Kaplan.

Moro F., and Amidei, B. **Relation between corneal degeneration of the "crocodile skin" type, and band shaped keratitis.** Gior. ital. oftal. **4**:444-464, Sept.-Oct., 1953.

A case of progressive, symmetrical and bilateral corneal degeneration of the cornea of the crocodile skin type (Vogt), in a myope, 70 years of age, is described in detail. The hystologic examination of a disc of cornea, removed from the opaque part, showed characteristics not unlike those observed in the early stage of band-shaped keratitis; this finding led the authors to suggest the hypothesis that the two conditions are different stages of the same abnormality. The possible part played by local vascular changes and alterations in the nerve supply in the causation of these conditions is discussed. V. Tabone.

Pines, N. **Denig's operation for trachomatous panus.** Brit. J. Ophth. **38**:186-187, March, 1954.

Lip mucus membrane had been grafted onto a trachomatous pannus by Denig's technique 22 years before. The graft remained in good condition and definitely remained lip membrane. The pannus was apparently cured. It is not known whether the graft serves as a mechanical protection between the rough lid and the sensitive cornea or the deep peritomy which is part of the operation creates a deep scar tissue barrier. (6 references) Morris Kaplan.

Poleff, L. **The present experimental state of the trachoma problem.** Arch. f. Ophth. **154**:381-388, 1953.

The author has studied the trachoma problem for many years and summarizes his findings as follows: Prowazek's inclusion bodies must be considered as representing the trachoma virus. The virus can be isolated and cultivated, it can be transmitted from tissue cultures to men and monkeys and can reproduce trachoma. Immunization against trachoma seems to be possible. Sulfa drugs and antibiotics are most valuable therapeutic agents. (21 references) Ernst Schmerl.

Rycroft, B. W. **The scope of corneal grafting.** Brit. J. Ophth. **38**:1-9, Jan., 1954.

The first record of the idea of corneal grafting to appear in the English language was probably made by Charles Darwin, who felt that the operation should be successful; since that time the idea and the operation have made tremendous strides and today promise hope of vision to many who are blind. Despite a reported successful case of animal cornea grafting in man, it was only after

principles of homoplasty were established that the technique developed rapidly. Now with the availability of precision instruments and strict asepsis, the operation is being performed in mushrooming numbers. It is now safe to say that in properly selected cases full-thickness grafts bring good results in 60 percent of cases, while lamellar grafts bring benefit in 80 percent.

The present-day problems fall under the classifications of technique, administration, and biological problems. In technique there still exists much discussion over the choice of direct suturing or a type of overlay suture; in the author's eye-bank the overlay suture is preferred. A careful consideration between full-thickness graft and lamellar graft should be made, and if some normal stroma overlies Descemet's membrane, the partial graft should be done. A full-thickness graft should not be done if binocular vision is better than 6/36 and should not be done if one eye is normal. Donor material is very difficult to obtain and should be well cared for; it should be cultured immediately and again before use. In about 10 percent of cadaver eyes after several days' refrigeration at 4°C. pathologic organisms have been cultured. The eyes may well be preserved in a mixture of paraffin, streptomycin and penicillin solution for as long as three weeks. Antibody reactions can and do occur, but are infrequent. It is hoped that full corneal grafts with some attached sclera will be successfully grafted in the future. (figures, 19 references)

Morris Kaplan.

Schirmer, R. **Caterpillar hair in the cornea.** Klin. Monatsbl. f. Augenh. **124**: 202-205, 1954.

The migration of these hairs toward the limbus could be observed. The active movements are explained by the special structure of these hairs and only few of them were surrounded by an infiltrate. (4 figures, 4 references)

Frederick C. Blodi.

Taborisky, J. **The pathology of trachoma.** Rev. intern. du trachome **31**:141-152, 1954.

In a study of the pathology of chronic trachoma in children from the third month after onset to final healing, the author found that cicatrization of the conjunctiva did not occur early in the disease. He was unable, even in severe cases, to demonstrate any trace of cicatrization before the third year of the disease. On the other hand, scar tissue was definitely present in cases of six years' duration. In his study he paid particular attention to epithelial alterations and noted a change, which began about the third or fourth day, from the normal regular cylindrical epithelium of two or three layers to a flat epithelium of one or two layers. The surface cells showed degenerative signs, from deterioration of the nuclear membrane to complete dissolution of the nuclear substance. In the subepithelial tissues there were signs of inflammation and edema, new vessel formation, follicles, and lymphocytic and plasma cell infiltration. In a similar manner the author analyzed the pathology of trachoma at two other periods. In the second period (trachoma from two to four years after onset) he observed multiplication of fibroblasts and their transformation into collagen fibers, as well as thickening and collagenization of reticular fibers. In the fourth and fifth years actual scar formation developed. The epithelium in succeeding years showed epidermoid changes, more marked in the tarsal areas than in the fornices. Fatty infiltration of the juxtatarsal tissues was found to be associated with atrophy of Mueller's muscle and involvement of the levator palpebrarum.

P. Thygeson.

Thygeson, Phillips. **Observations on nontuberculous phlyctenular keratoconjunctivitis.** Tr. Am. Acad. Ophth. **58**:128-132, Jan.-Feb., 1954.

Although allergy to tuberculoprotein is considered the most important cause of phlyctenular keratoconjunctivitis, rare cases appear to be due to allergies to other bacterial, viral or fungal proteins. Staphyloccus aureus provided the most important of these other sensitizing proteins in 37 cases of phlyctenulosis seen in the San Francisco Bay area over a four-year period. In five cases evidence of the relationship of the disease to sensitivity to staphylococcus proteins was deduced from the patient's favorable response to antistaphylococcic topical therapy, from the patient's extremely high sensitivity to staphylococcus toxoid, and from the fact that the disease recurred only when the staphylococcic lid infection recurred. Phlyctenulosis, apparently due to sensitivity to Candida albicans (monilia) (1 case), to lymphogranuloma venereum virus (1 case), and to Coccidioides immitis (2 cases), was also observed. (1 table, 7 references) Theodore M. Shapira.

Torfeh, H. **The effect of gantrisin in the treatment of trachoma.** Rev. intern. du trachome **31**:78-82, 1954.

The author reports that gantrisin was well tolerated by mouth or intramuscular injection and that good blood levels were obtained with the standard dosage. In a therapeutic study of 260 cases of trachoma, gantrisin was found to be specially effective in Stage I, but in Stages II and III treatment times of two months or longer were required to effect cures. The effect of gantrisin on secondary infections was striking. Inclusions were present in 30 percent of the cases before treatment; after two weeks of treatment all cases had become inclusion-free. Provocative tests with cortisone did not induce a return of inclusions. P. Thygeson.

Toulant, P., and Boithias, M. **Herbert's peripheral pits in Negroes.** Rev. intern. du trachome **31**:75-77, 1954.

The authors have observed in trachomatous Negroes a characteristic pigmentation of Herbert's pits. In place of the usual transparent depth, they noted a heavily pigmented layer. Under the biomicroscope the pigment was seen to be localized in the superficial layer of the epithelium. The diagnostic value of these pigmented pits is stressed. The authors consider that trachoma, though mild in the Negro, is fairly common with an index of 20 to 30 percent in French West Africa.
P. Thygeson.

Ueoka, T., and Ito, N. **So-called acute spring catarrh.** Acta Soc. Ophth. Japan **58**:346-352, April, 1954.

Oguchi and his collaborators described "acute spring catarrh" a few years ago and claimed an acute onset. The present authors reexamined the problem. They observed 20 such cases, and agreed with the conception that the onset of spring catarrh is acute, although it follows a chronic course. (3 figures, 1 table, 8 references) Yukihiko Mitsui.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Alagna, G. **Gyrate atrophy of choroid and retina (clinical and pathogenetic contribution).** Arch. di ottal. **58**:33-65, Jan.-Feb., 1954.

After a review of the literature the author describes a case and discusses the pathogenesis and the differential diagnosis. Gyrate atrophy of choroid and retina is a well defined entity and can be differentiated from pigment degeneration or diffuse primary sclerosis of the choroidal vessels. True choroideremia and congenital total coloboma of the choroid represent identical congenital defects, while the

partial or total absence of the choroid in gyrate atrophy is merely an aspect of the primary degenerative process in the choroid. The cause is probably a congenital lability of the choroidal vascular system. (2 figures, 81 references)

John J. Stern.

Calhoun, F. P. **Diseases of the uveal tract.** A.M.A. Arch. Ophth. **51**:376-399, March, 1954.

This excellent review of 190 articles on diseases of the uveal tract covers the important available papers which were published between October, 1952 and October, 1953. (190 references)

R. W. Danielson.

Fair, John R. **Uveitis: a military problem.** A.M.A. Arch. Ophth. **51**:364-368, March, 1954.

A report of 97 cases of uveitis is presented in which the Sabin-Feldman dye test was used. The sera from only nine of these cases were reported as positive in a dilution of 1:256 or higher. The authors stress that what is needed is a combined serologic and pathologic study, with isolation of the organism, before acquired ocular toxoplasmosis can be proved or disproved. They believe that leptospiral uveitis is not a common cause of uveitis in military personnel. (2 figures, 2 tables, 7 references) R. W. Danielson.

François, Jules. **Fuchs's heterochromia.** Ann. d'ocul. **187**:255-272, March, 1954.

The chief manifestations of Fuchs's heterochromia are hypochromia of the iris, atrophy of the iris, keratic precipitates, cataract, disturbances of the vitreous with the presence of white spots in the anterior layers, and the absence of all signs of inflammation including posterior synechiae. Gonioscopic examination is often negative. Sometimes small vascular branches are seen along the base of the iris, and at other times radial vessels which run from the angle as far as the level of the spur or Schwalbe's ring.

The filiform hemorrhage which follows anterior chamber puncture is pathognomonic of the Fuchs syndrome. It may occur in any part of the angle and sometimes simultaneously at different points. It originates from the canal of Schlemm. Examination of the aqueous generally shows a positive Pandy test, but few or no cellular elements.

The syndrome is sometimes, but only rarely, bilateral, and the bilaterality is present from the outset. The prognosis depends mainly on the presence or absense of glaucoma, which appears in 4 to 5 percent of cases, and more frequently in the bilateral than in the unilateral cases.

Histopathological examination shows few changes, degenerative rather than inflammatory in nature. Tests with cocaine, adrenaline, and benzedrine suggest a paresis of the sympathetic. Treatment is only necessary if cataract or glaucoma is present. (7 figures, 21 references)

John C. Locke.

Fukuda, M. **Keto-enol substance in the urine of sympathetic ophthalmia patients.** Acta Soc. Ophth. Japan **58**:188-190, Feb., 1954.

Keto-enol substance in the urine was measured in three cases of sympathetic ophthalmia. An enormous increase was observed in the convalescent stage. Cortisone impeded the increase of the substance. The author considers the increase to be due to an abnormal protein metabolism in the pigmented cells. (2 figures, 1 reference) Yukihiko Mitsui.

Hudson, J. R. **Toxoplasmic choroido-retinitis in the adult.** Brit. J. Ophth. **38**: 179-181, March, 1954.

Hudson describes a case of acute choroido-retinitis in a 35-year-old man with no previous history of eye disease. He re-

acted positively to the dye test and also to the complement-fixation tests though only to a very low titre. He was treated by cortisone and atropine and apparently healed well although after healing his test titre rose to 1:800 without adequate explanation. He has remained well healed after one year of observation. (1 figure, 1 table, 6 references) Morris Kaplan.

Jacobs, L., Cook, M., and Wilder, H. **Serologic data on adults with histologically diagnosed toxoplasmic chorioretinitis.** Tr. Am. Acad. Ophth. **58**:193-200, March-April, 1954.

This discussion is based on a small number of cases, in which statistically highly significant results have been obtained. The ultimate demonstration of the toxoplasmic etiology of uveitis must come by the isolation and identification of parasites in typical cases. Until such evidence is obtained, the question of the significance of the dye test for toxoplasmosis in the diagnosis of ocular disease will not be resolved. However, on the basis of the author's present thesis it is suggested that aside from rising titers the only dye test results which can be considered really significant are those which are completely negative. Those which are very low, such as undiluted or 1:4, may be of no significance in the presence of active lesions. Those which are very high, consistently over 1:1,000, may be indicative of toxoplasmosis. Intermediate titers merely indicate that toxoplasmosis may be considered in the differential diagnosis, but there appears no basis for the postulation that titers of 1:256 are more definitely diagnostic than lower values. The data on the complement fixation test in the group of patients reported here indicate that this test is of no value in the diagnosis of old infections. It does not appear likely that serologic testing will furnish an easy method for diagnosis of toxoplasmic chorioretinitis; it will provide merely one other datum for the ophthalmologist to consider in his complete medical survey of the individual patient. (1 table, 16 references)

Theodore M. Shapira.

Kimura, S. J., Hogan, M. J., and Thygeson, P. **Uveitis in children.** A.M.A. Arch. Ophth. **51**:80-88, Jan., 1954.

This is a survey study of 810 cases of uveitis. 47 occurred in children under 16 years of age. Extensive clinical and laboratory tests were conducted on each case. 18 cases showed anterior uveitis and 29 posterior uveitis. Etiologic factors were found in 6 of the 18 cases of iridocyclitis. These were heterochromic iridocyclitis, sarcoiditis, lues, trauma, brucellosis, and juvenile rheumatoid arthritis. A presumptive diagnosis of toxoplasmosis was made in 10 of the cases of posterior uveitis. 19 were unexplained. A definite diagnosis of toxoplasmosis is difficult after the age of 5 years because of low titers. The authors point out that subsequent siblings of children with toxoplasmosis are likely to be toxoplasmosis-free (8 tables, 27 references) G. S. Tyner.

Leopold, I., and Dickinson, T. **Antihyaluronidase and antistreptolysin titers in uveitis.** Tr. Am. Acad. Ophth. **58**:201-211, March-April, 1954.

Sixty-three cases of uveitis were studied for serum levels of antihyaluronidase and antistreptolysin. The increase in geometric mean titer, the shift to the right of the frequency distribution of titers as compared to the normal, and the IRD of 20 percent above the minimal useful point all suggest that the cases of uveitis in the study do not represent a normal group with regard to serum antihyaluronidase and antistreptolysin. The data presented here suggest that the hemolytic streptococcus may play an etiologic role in the production of uveitis; also that these tests, particularly the antihyaluronidase titer,

should be studied further as a possible method of detecting the etiology of uveitis. (8 tables, 23 references)

Theodore M. Shapira.

Leydhecker, W. **Myopia, uveitis, debility and hydrocephalus in identical twins.** Klin. Monatsbl. f. Augenh. **124**: 306-309, 1954.

These six-year-old girls were assumed to be homozygous. They showed these numerous pathologic findings without any signs (radiologic or serologic) of toxoplasmosis. The author believes that this is an instance of the so-called Sabin-Feldman syndrome. (17 references)

Frederick C. Blodi.

Médinger, F. **A case of Harada's disease.** Arch. d'opht. **14**:172-174, 1954.

The author describes the first reported Algerian case of Harada's disease which occurred in a woman of 27 years without previous ocular disease. The initial symptom was a veil-like disturbance of vision of the right eye, with visual acuity of 3/10 in this eye and 10/10 in the left. The fundus picture consisted in edema of the retina, whitish-gray exudates around the disc, hyperemia of the disc, and tortuosity of the vessels. Headache of increasing intensity was the only initial general symptom but this was soon followed by fever and nervous unrest. On the sixth day the left eye began to fail, showing the same retinal changes as had appeared in the right eye. By the tenth day vision had been reduced to light perception and the general condition had worsened, with fever, headache, and neck rigidity. A lumbar puncture showed a lymphocytic reaction. This was followed by an increase in the ocular signs eventuating in total retinal detachment, with light perception only, while at the same time the systemic symptoms gradually disappeared. The anterior segment was never involved. After four months vision had returned to R. 2/10, L. 1/10, and the detachment had subsided. After two years the vision had returned to 9/10 in each eye and the discs and vessels were apparently normal. In reviewing the literature on Harada's disease, Médinger notes the frequent occurrence of skin and hair changes. Their absence in his case does not in his opinion negate the diagnosis in view of the typicality of the uveal and nervous signs.

P. Thygeson.

Pietruschka, Georg. **Clinical observations in iridocyclitis.** Klin. Monatsbl. f. Augenh. **124**:309-326, 1954.

The fundus of 45 patients with acute iridocyclitis was observed and followed; 17 patients showed marked fundus changes. Dilatation of the retinal veins was the most common finding. Edema of the disc or the macula or both occurred The picture of a papilledema was also simulated. Cellular infiltrates in the vitreous were frequent. (4 figures, 2 charts, 56 references) Frederick C. Blodi.

Rosetti, D., and Roi, G. **A case of heterochromia of the iris and hemifacial hyperhydrosis.** Ann. di ottal. e clin. ocul. **79**:11-22, Jan., 1953.

A girl, 9 years of age, exhibited the combination described in the title. The authors suspect a lesion of the sympathetic fibers in contact with the fifth cranial nerve, acquired either during intrauterine life or during delivery by forceps. The type of heterochromia described by Fuchs can be excluded because of the absence of inflammatory signs or cataract. A miosis in the affected eye, pharmacological tests on the pupil and the hemihyperhydrosis point toward a sympathetic paresis. (1 figure, 80 references) John J. Stern.

Scheie, Harold G. **Gonioscopy in diagnosis of tumors of iris and ciliary body.** A.M.A. Arch. Ophth. **51**:288-300, March, 1954.

No mention has been made in the literature of the fact that the extreme periphery of the posterior chamber of many eyes can be seen by gonioscopic examination with the pupils dilated. This paper discusses the various lesions that can be so observed, with special reference to intraepithelial cysts, the commonest space-taking lesion of the posterior chamber.

The gonioscopic characteristics of the cystic lesions observed in the patients reported in this paper bear out the microscopic observations of Reese. Those of the ciliary body were transparent because they were covered by transparent epithelium, whereas those at the periphery of the iris were opaque and melanotic in appearance. This type of examination has its greatest value in the diagnosis of tumors of the posterior chamber whose only presenting symptom may be a bulge of the periphery of the iris. (12 figures, 1 table, 4 references) R. W. Danielson.

Unger, H., and Schomerus, E. **Bandkeratopathy in infantile iridocyclitis.** Klin. Monatsbl. f. Augenh. **124**:326-334, 1954.

Among 19 children with iritis and bandkeratopathy only nine developed arthritis. These ocular signs are therefore not necessary pathognomonic for Still's disease. The author believes that tuberculosis should still be considered as a possible etiologic factor. (1 figure, 43 references) Frederick C. Blodi.

Vannini, Angelo. **Gonioscopy in heterochromia iridis.** Rassegna ital. d'ottal. **23**: 3-23, Jan.-Feb., 1954.

Vannini presents his findings in the study of the anterior chamber in 18 cases of heterochromia of the iris, often called the syndrome of Fuchs. Three different pictures were seen with the gonioscope, 1. normal angles, 2. blood vessels of notable size in the angle of the chamber, and 3. an anomaly of congenital origin in which the blood vessels may also be numerous. The anomaly consists of an embryonic, undifferentiated mesodermic tissue, rich in capillaries. Of the 15 cases described, eight showed a normal angle of the anterior chamber, three presented numerous blood vessels and four exhibited the congenital defect described. (10 figures, 53 references) Eugene M. Blake.

Wager, H. E., and Calhoun, F. P. Jr., **Torula uveitis.** Tr. Am. Acad. Ophth. **58**:61-67, Jan.-Feb., 1954.

The authors describe the fourth recorded instance of intraocular infection by Torula histolytica (Cryptococcus neoformans). The general and ocular manifestations were variable in these four cases, but all patients had signs of a disturbance of the central nervous system and all died. Three of the patients had proven meningitis, but the cause of death was unknown in the fourth. One patient with meningitis also had malignant lymphoma. Apparently the intraocular disturbance is always a manifestation of a generalized cryptococcal infection which may or may not have been diagnosed clinically. It is suggested by the authors that the possibility of a torular infection should be considered when patients with uveitis, Hodgkin's disease or other lymphoma exhibit signs of disease of the central nervous system. (5 figures, 7 references) Theodore M. Shapira.

Woods, Z., Jacobs, L., Wood, R., and Cook, M. **A study of the role of toxoplasmosis in adult chorioretinitis.** Tr. Am. Acad. Ophth. **58**:172-192, March-April, 1954.

The study has yielded considerable circumstantial evidence that adult acquired ocular toxoplasmosis is a clinical entity. In this series of 201 cases of granulomatous uveitis 58 can reasonably be ascribed to Toxoplasma; in 17 of these patients the infection was probably acquired congenitally, and in 41 it probably occurred in

adult life. Toxoplasma infection is the probable etiologic factor in approximately 25 percent of adult granulomatous uveal disease. The diagnosis of the Toxoplasma etiology can only be made by a careful consideration of the clinical ocular picture, an evaluation of the results of a careful medical survey, and the results of the serologic and cutaneous Toxoplasma tests. In the evaluation of a single positive dye test as an index of a Toxoplasma etiology, there appears to be a 20 to 25 percent margin of error. (8 tables, 25 references) Theodore M. Shapira.

Zarrabi. **A case of bilateral iritis with poliosis.** Arch. d'opht. **13**:776-778, 1953.

This is a report of a bilateral iritis in a 51-year-old man which followed two months after extracapsular cataract extraction of the right lens. The iritis started first in the unoperated eye, and then developed in the aphakic eye. There was a low fever of short duration. A few weeks after onset, beginning poliosis involving the scalp, eyebrows, and cilia was detected. The uveitis evolved in three exacerbations during each of which an increase in the poliosis was noted. Treatment with cortisone, aureomycin, and fever therapy failed to affect the disease which was still active when the patient was last seen.

The author discusses the case in relation to sympathetic ophthalmia and to the Vogt-Koyanagi syndrome. (1 figure) P. Thygeson.

9

GLAUCOMA AND OCULAR TENSION

Bain, W. E. S. **Variations in the episcleral venous pressure in relation to glaucoma.** Brit. J. Ophth. **38**:129-135, March, 1954.

Variations in episcleral venous pressure in relation to glaucoma were first studied by Thomassen. In this study the work is confirmed and elaborated upon. The instrument consisted of a very thin rubber diaphragm connected to a viewing chamber which in turn was connected to a well regulated bellows. The diaphragm was placed against the lateral aspect of the sclera while simultaneous tracings were made of venous and of intraocular pressures. In normal as well as in glaucomatous eyes it was found that there is a direct relationship between the two pressures and that the changes in venous pressure definitely preceded changes in the intraocular pressure, both in elevation and return to a base level. These changes were generally bilateral even when only one eye was glaucomatous. After the instillation of a miotic in either simple or congestive glaucoma the venous pressure was reduced first and the intraocular pressure at a later stage. It is noteworthy that the changes in the venous pressure were more marked and more immediate after eserine than after pilocarpine (about 5 minutes with the former and 40 minutes with the latter). (14 figures, 7 references) Morris Kaplan.

Caselli, Francesco. **The Findeisen test in glaucoma.** Arch. di ottal. **58**:67-74, Jan.-Feb., 1954.

The Findeisen test is a combination of the Schellong test and the Braasch test. The former consists in a comparison of the systolic arterial pressure in the upright and the horizontal position: a fall of 15 mm. Hg or more is considered positive. The latter test consists in a diminution of the pulse rate on flexing the neck so that the chin touches the sternum; a slowing down of eight or more beats is positive. The combination of both tests demonstrates the degree of dystonia of the neurovegetative system which is the foremost predisposing factor in glaucoma. Of 58 patients examined with Findeisen's test 80 percent were found to show a positive result. This test is valuable in demon-

strating a predisposition for glaucoma in doubtful cases. (1 table, 26 references)

John J. Stern.

Colombi, Carlo. **Cortisone and ocular tonus.** Rassegna ital. d'ottal. **23**:63-67, Jan.-Feb., 1954.

The author studied two groups of 20 subjects, each of whom received six daily instillations of 1-percent cortisone. He measured the pressure before treatment and one hour and three hours after the last instillation. The first group was composed of young individuals, aged 5 to 30 years of age and treated for various conditions such as strabismus, central scotoma, contusion of the globe, angioma of the eyelid, orbital sebaceous cyst and posterior cortical cataract. The second group consisted of older patients, aged from 57 to 85 years. They had senile cataracts, myopic cataract and dacryocystitis. Every one of the 40 subjects examined presented a normal tonus, almost identical in the two eyes. The average variation in tonus was 4 to 5 mm. None of the three subjects with chronic simple glaucoma and four with inflammatory glaucoma, showed a change of more than 4 to 5 mm. of pressure. The same result was found in treating a patient with a compensated bilateral hydrophthalmos with cataract, and one with heterochromia iridis of Fuchs for three days. (5 references)

Eugene M. Blake.

Huerkamp, B., and Pohle, H. **The intraocular pressure during the second half of the night.** Klin. Monatsbl. f. Augenh. **124**:296-304, 1954.

For four years all hospitalized patients with glaucoma had the intraocular pressure measured twice during the day and once during the night. In one series of 100 patients the nocturnal tension was measured at 1 a.m., in a second series of 100 patients at 2 a.m., and so on until 7 a.m. All these measurements were repeated for an average of 15 days. Nearly 75 percent of the eyes measured showed a falling pressure curve. Only at 6 a.m., that is one hour after rising, was the curve reversed and this tendency reached a peak at 7 a.m. (2 charts, 22 references)

Frederick C. Blodi.

Huerkamp, B., and Zieglitz, W. **Ozone content of the air and glaucoma.** Arch. f. Ophth. **154**:507-515, 1953.

For about three months the changes in ocular tension of 71 patients with chronic glaucoma and changes of the ozone content of the air were recorded. About 90 percent of the patients showed no definite correlations. (2 figures, 35 references)

Ernst Schmerl.

Magitot, A. **Ocular hypertension is a symptom and not a disease.** Ann. d'ocul. **187**:1-24, Jan., 1954.

The author maintains that hypertension and glaucoma are two different things. However, all ocular hypertensions, whether they are glaucomatous or not, result from the same mechanism, namely, engorgement of uveal capillaries and hypersecretion of intraocular fluid. He feels that every ocular hypertension is secondary to increased venous pressure and is analogous to edema of other organs.

Most manifestations of glaucoma are not caused by increased intraocular pressure. The excavation of the optic disc is vascular in origin, and the field changes are not pathognomonic. Provocative tests are not consistent in their results, because they depend on the neuropsychiatric state of the patient, which varies from day to day. In evaluating results of surgical treatment, the functional state of the eye, rather than the ocular tension, should be the criterion used. (10 references)

John C. Locke.

Sanna, M. **Effect of nicotine on intraocular pressure and on the angioscotoma**

of the glaucomatous eye. Boll d'ocul. **33**: 31-39, Jan., 1954.

The ocular tension and the angioscotoma were studied in 25 glaucomatous patients, before smoking a cigarette and 5, 15, 25, and 60 minutes after. All tests were performed in the early afternoon hours and the patients had no access to tobacco before the test cigarette. In eyes with mild hypertension the nicotine inhalation produced a temporary increase in pressure, accompanied by an enlargement of the angioscotoma; after one hour values similar to those preceding the test were found. (Two tables) In eyes with higher initial pressure readings (eight out of 25 in this series) angioscotoma remained increased for a longer time. Smoking may become an additional factor for deterioration of a glaucomatous eye. (2 tables, 15 references) K. W. Ascher.

Sedan, Jean. **Individual and familial factors in the slow obliteration of antiglaucomatous fistulas.** Ann. d'ocul. **187**:31-39, Jan., 1954.

The author presents observations (bilaterality of closings in the same person, closings in several members of the same family) suggesting that constitutional and familial factors predispose to the slow obliteration of previously functioning fistulas. Hypercholesterolemia may be a contributing factor. (12 references)

John C. Locke.

Shaffer, Robert N. **The role of vitreous detachment in aphakic and malignant glaucoma.** Tr. Am. Acad. Ophth. **58**:217-231, March-April, 1954.

Vitreous detachment is present in almost all eyes which have had intraocular surgery, and in many eyes suffering from glaucoma. In some aphakic eyes and in eyes with subluxated lenses, synechias can form between iris and the vitreous face producing a pupillary block. Aqueous collects in the posterior chamber, detaching the vitreous anteriorly, or goes behind a posteriorly detached vitreous body, shoving it forward, producing a glaucoma. The anterior chamber can be either collapsed or filled with vitreous. Iridectomy will usually result in a cure of this type of glaucoma.

A similar syndrome is sometimes seen after lens extraction is done to relieve a malignant glaucoma. It becomes impossible to refill the anterior chamber because aqueous runs posteriorly, forcing detached vitreous and iris forward and continuing the malignant course. Only by breaking the pupillary block can this type of glaucoma be relieved. If the aqueous is collecting anteriorly, iridectomy and lens extraction will be helpful. If aqueous is collecting behind the detached vitreous, the only chance of cure is in actual incision through the detached vitreous to break the postero-anterior pupillary block. (7 figures, 12 references)

Theodore M. Shapira.

Smith, Redmond. **Gonioscopic studies in congestive glaucoma.** Brit. J. Ophth. **38**:136-143, March, 1954.

Gonioscopic studies of 13 cases of congestive glaucoma were made during various phases of the attack before and after instillation of a miotic; all presented unquestioned narrow filtration angles. There seemed to be no appreciable correlation between the amount of obstruction of the angle and the extent of the rise in pressure. However, if the light of the slit-lamp was turned off and on during the examination, the angle was seen to open and close. Without this technique most of the angles appeared more or less open because of the bright light, but those angles which remained open when the light was turned off gave much more of an insight into the prognosis of the disease. It is recommended that this "off-on" technique be explored more widely. (1 table, 17 references) Morris Kaplan.

Stambaugh, J. L., Fuhs, J. C., and Ascher, K. W. **Study of the compensation-maximum test on aqueous veins.** A.M.A. Arch. Ophth. **51**:24-31, Jan., 1954.

During compression of the globe the dynamometer pressure necessary to produce a retrograde influx of blood from episcleral veins into aqueous veins has been termed the "compensation maximum." Glaucomatous eyes require much less pressure than normal eyes to produce this phenomenon. The authors believe this test is not "—sufficiently correlated or characteristic to be of value in clinical differentiation of these two groups." (2 figures, 2 tables, 19 references)

G. S. Tyner.

Urrets-Zavalia, A., Jr. **Etiopathogenesis of chronic simple glaucoma.** Arch. d'opht. **13**:749-772, 1953.

The author considers the pathogenesis of chronic simple glaucoma in the light of recent investigations, particularly as related to stress phenomena and to tonography. He considers the dual clinical picture of the disease to correspond correctly to its dual pathogenesis. In chronic simple glaucoma the iris angle is always open, whether ample or narrowed, and a resistance to outflow of aqueous is explained by various changes occurring in the angle, such as diffuse sclerosis and hyaline degeneration, or by increase in pressure in the intrascleral venous plexus, all of which are associated with a sclerosis of the capillary network in the anterior uvea. He further considers that a similar sclerosis localized in the capillary network on the circle of Haller and in the retinal vascular tree is the cause of the lacunar degeneration of the optic nerve and of alterations of the visual field. He concludes that this capillary sclerosis is manifested not only in the eye but can be demonstrated also in other parts of the body. The relationship of glaucoma to the adaption syndrome of Selye is considered. (3 figures, 67 references) P. Thygeson.

10

CRYSTALLINE LENS

Auricchio, G., and Ambrosio, A. **Studies on the pathogenesis of complicated cataract, following iridocyclitis.** Gior. ital. oftal. **4**:489-493, Sept.-Oct., 1953

This is the fourth article on studies of complicated cataract. The authors have determined the contents of pyruvic and lactic acid in the aqueous, vitreous and lens, after experimentally produced tuberculous uveitis. They found that these substances become diminished in the intra-ocular fluids and increased in the lens substances under these conditions

V. Tabone.

Cameron, A. J. **Experience with the Harrington erisophake in fifty cataract extractions.** Brit. J. Ophth. **38**:173-178, March, 1954.

The Harrington erisophake is a modification of the original Dimitry and later the Bell erisophake in that the pressure is supplied by a rubber bulb contained within a metal tube of about the shape and size of an ordinary pencil. The use of this instrument in 50 unselected cases of cataract is described in which the results generally were as good or better than those obtained from the classical methods of extraction. It is stressed, however, that it is not recommended that this method supplant the standard extracapsular extraction. (2 tables, 9 references)

Morris Kaplan.

Gardella, Giovanni. **Calcareous posttraumatic cataract.** Minerva med. **2**:1874-1876, Dec. 15, 1953.

Gardella describes posttraumatic calcification and opacification of the lens which was demonstrable roentgenographically. He discusses the conditions in which

calcification can develop and reviews the literature. (4 figures, 12 references)

F. H. Haessler.

Hallerman, W., and Meisner, G. **The debatable tumor immunity of the lens.** Klin. Monatsbl. f. Augenh. **124**:159-164, 1954.

In a case of malignant melanoma of the ciliary body and the anterior choroid a nest of tumor cells was found histologically in the lens. This was interpreted as an intraocular metastasis and would speak against the absolute tumor immunity of the lens. (6 figures, 5 references)

Frederick C. Blodi.

Legrand, J. **Cataracts and antiglaucoma operations.** Ann. d'ocul. **187**:25-30, Jan., 1954.

The author believes that the frequency of cataracts after fistulizing operations is the result of changes in the position of the lens and the composition of the aqueous humor.

John C. Locke.

Meesmann, A., and Lange, F. **Pupillary contraction during cataract surgery.** Arch. f. Ophth. **154**:491-506, 1953.

The authors report on 175 cases of surgery for cataract. They recommend the use of glaucosan or homatropine before surgery and the rinsing of the anterior chamber with a solution of ½ percent of pilocarpine after surgery. Complications after surgery were found more frequently where the peripheral iridectomy had been omitted, and it seems that the latter procedure is always indicated. (5 tables, 15 references)

Ernst Schmerl.

Pau, Hans. **The permeability cataract.** Klin. Monatsbl. f. Augenh. **124**:1-29 and 129-148, 1954.

The author gives first a short survey on some problems of lens metabolism. He reports his own histochemical studies. Formazan crystals are formed near the capsule and around the sutures. They are probably produced by dehydrases. The pupillary area is relatively free from this enzyme and this distribution corresponds to the deposition of silver or copper in the lens. The diffuse deposition of iron beneath the anterior capsule corresponds more to the distribution of the cytochrome oxidase system.

A functional "membrane" consisting of capsule, epithelium and superficial stroma has a selective permeability. When this membrane is damaged, for example, after death, a loss of potassium occurs and the weight of the lens decreases. Later sodium is taken up by the lens and the weight of the lens increases. Similar changes occur in a cataractous lens. Most cataracts are permeability cataracts in which a disturbance of the normal permeability of this functional membrane leads to an exchange of electrolytes and an uptake of water. Among the permeability cataracts, the author discusses endocrine cataracts (diabetes, tetany), toxic cataracts (thallium, naphthalene), irradiation cataracts and cataracts secondary to intraocular diseases. The treatment with cystein should be of some value.

Frederick C. Blodi.

Reeh, M., and Lehman, W. **Marfan's syndrome (arachnodactyly) with ectopia lentis.** Tr. Am. Acad. Ophth. **58**:212-216, March-April, 1954.

A white man, 25 years of age, died shortly after admission to the hospital to which he had been admitted for pain which was caused by a dissecting aneurysm of the aorta. A diagnosis of Marfan's syndrome had been made at the ophthalmology clinic of the Oregon medical school when the subject was 11 years old. The eyes were removed at autopsy and are described in detail in this report. It is emphasized that in Marfan's syndrome

many abnormalities are present which make ocular surgery hazardous. The sclera may be so thin that one may completely perforate the globe when making a shallow incision for limbal sutures. Abnormalities in the iris make it necessary to do an iridectomy before delivery of a dislocated lens. The poorly developed ciliary body may produce aqueous inadequately after surgery, and severe hypotony and even ultimate loss of the eye may result. (3 figures, 1 reference)

Theodore M. Shapira.

Reese, A. B., and Wadsworth, J. A. C. **Occurrence of cystoid spaces in the lens.** A.M.A. Arch. Ophth. **51**:315-317, March, 1954.

The authors report the presence of cystoid spaces in the lens in about 10 percent of patients over 40 years of age. They occur just beneath the anterior and posterior surfaces of the adult nucleus. With the ophthalmoscope the spaces beneath the anterior surface of the adult nucleus are seen as small, oval to round, grayish opacities arranged in a more or less symmetric fashion with the long axis concentric to the visual axis. When the optical beam of the biomicroscope is adjusted to the anterior surface of the adult nucleus, the lesions bulge forward; when focused deeper, the lesions appear as empty cystoid spaces with some increase in density of the walls.

These lesions are not to be confused with the ordinary vacuoles, which are usually arranged in a rather irregular fashion in the cortex and even in the subcapsular area. No references to them were found in the literature. (2 figures, 1 reference)

R. W. Danielson.

Rese, Warren S. **Reports on the use of the intraocular acrylic lens (Ridley operation).** Tr. Am. Acad. Ophth. **58**:55-60, Jan.-Feb., 1954.

Of the 11 implantations attempted, two were abandoned at surgery because of loss of vitreous, two lenses were removed because of severe iritis and glaucoma and in one of the patients there was a question of sympathetic ophthalmia. Three patients developed an occluded pupil and one of these ended in discission and unintentional dislocation of the acrylic lens. In four patients the visual acuity, with optical correction, is 20/40 or better and two have binocular single vision with fusion as shown by the Worth four-dot test. All patients had varying degrees of iritis and in most of them adequate postoperative funduscopic examination was impossible.

Theodore M. Shapira.

Ridley, Harold. **Further experiences of intra-ocular acrylic lens surgery.** Brit. J. Ophth. **38**:156-162, March, 1954.

The author now reports his experience in the use of the acrylic lens implant during cataract surgery in 150 cases during a three and one half-year period. He has changed the technique only slightly. Cocaine and adrenaline are used before surgery rather than homatropine and intracapsular extraction of the lens has been abandoned since the implant is prone to become dislocated into the vitreous after trauma. The visual results continued to be good or better than at first and complications have become less. The operation is recommended for all cases of monocular cataract and it is suggested that it be given consideration in routine cataract surgery. (3 tables, 17 references)

Morris Kaplan.

11

RETINA AND VITREOUS

Ashton, Norman. **Animal experiments in retrolental fibroplasia.** Tr. Am. Acad. Ophth. **58**:51-54, Jan.-Feb., 1954.

The author states that it does not necessarily follow that abnormal retinal proliferations in the premature baby occur

exactly as in the kitten and it is more than probable that other factors are also involved, but he believes that high concentrations of oxygen, by obliterating the vessels and inducing retinal ischemia, are responsible for the majority of cases of retrolental fibroplasia, while those occasional cases which have not been subjected to oxygen therapy and the rare prenatal form of the disease may possibly be due to vascular aberrations consequent upon prolonged anoxia from fetal or maternal causes.

The experimental findings presented are sufficiently definite to indicate that the uncontrolled employment of oxygen is extremely dangerous in the treatment of premature babies, in that it is capable of destroying the normal process of retinal vascularization. The author expresses the hope that the experimental results may persuade clinicians to measure accurately, and at frequent intervals, the oxygen concentration within the baby incubators; to give oxygen only when indicated and then in the minimum quantity and for the shortest possible period consistent with the infant's survival. (9 references)

Theodore M. Shapira.

Bonavolontà, G. **Clinical considerations on some aspects of diathermic retinochoroiditis in detachment of the retina.** Gior. ital. oftal. **4**:409-422, Sept.-Oct., 1953.

The author reports the pathologic effects of diathermy in retinal detachment. The results in 50 cases are given in some detail; in 18 cases, the retina was approximated to the choroid at the time of operation, while in the other 32 cases it was well away from the choroid. In all cases in the former category, the retina was visibly coagulated at the time of application, while in the other cases, it usually became subsequently so, after drainage of the sub-retinal fluid. The destructive effects of diathermy on the retina were very much more marked in those cases in which the retina was approximated to the choroid, and the evolution of the whole process usually took three days longer. After a reminder of the frequency of a retinal detachment at the site of the treated region, and of its differential diagnosis from true idiopathic detachment, the author advances the hypothesis that the degenerated retina stimulates and prolongs the process of choroido-retinitis after diathermy; this may explain the exceptional cases of spontaneous reattachment, or sealing of retinal holes.

V. Tabone.

Bonnet, Paul. **Congenital tortuosity of the vessels of the retina and congenital stenosis of the aortic isthmus (coarctation of the aorta).** Arch. d'opht. **14**:129-139, 1954.

In 1950 the author described the frequent association between congenital tortuosity of the vessels of the retina and congenital stenosis of the isthmus of the aorta. In the present article he discusses in detail the clinical picture of the retinal disease and notes that three clinical types may be defined as follows: 1. Tortuosity involving the veins only (6 cases); 2. tortuosity involving the arteries only (21 cases); and 3. tortuosity involving both arteries and veins (9 cases). In a similar manner he defines the clinical characteristics of coarctation of the aorta and stresses that the most characteristic sign is the discordance between the circulatory state of the upper part of the body and that of the lower. The average survival rate has been around 35 years and death has resulted from rupture of the aorta, cardiac failure, intracranial hemorrhage, or infectious endocarditis or aortitis. Bonnet also discusses the effect of the operation of resection of the stenosis which now has a mortality rate of around 8.5 percent. He continues with a discussion of the relationship of the retinal changes to the hypertension and con-

cludes, in opposition to the opinions of Granstrom and of Walker and Stanfield, that the tortuosity is a congenital lesion which may be modified only slightly by the superimposed hypertension. (18 references) P. Thygeson.

Bossu, A., and Lambrechts. **Ocular manifestations of the Ehlers-Danlos syndrome.** Ann. d'ocul. **187**:227-236, March, 1954.

The three cardinal features of the Ehlers-Danlos syndrome are: hyperelasticity and fragility of the skin, and hypotonic ligaments. Ocular lesions are rare. They tend to be polymorphous, because of the fact that this is a degenerative disease of the entire mesenchymal system. The authors present the case of a 19-year-old girl who had, in the right eye, a detachment of the retina secondary to retinitis proliferans, and, in the left eye, beginning macular degeneration. It was thought that these lesions might be due to a series of small chorioretinal hemorrhages, even though hematologic examinations were negative. (5 figures, 37 references) John C. Locke.

Fontaine, M. **Retinopathy of premature infants.** Arch. d'opht. **14**:140-153, 1954.

This review of retrolental fibroplasia adequately describes the clinical features of the disease and is based on personal experience with 144 retinopathies representing 13 percent of 1,019 premature infants of less than 2,000 gm. birth weight. Of the 144 retinopathies, 90 regressed totally, 14 left partial sequelae with conservation of a certain amount of vision, and 40 progressed to complete fibroplasia with blindness. The period of evolution of the disease in the observed cases varied from four to 12 weeks. A few cases were noted in which, after total retinal detachment, reattachment took place although the visual prognosis was considered to be extremely poor. The author discusses the various treatments tried and notes their complete lack of success. In considering etiology he states that prematurity is the sole certain factor but that the role of oxygen and anoxia cannot be ignored in view of current researches. (30 references) P. Thygeson.

François, J., and Verriest, G. **Visual functions in retinal elastosis.** Ann. d'ocul. **187**:113-144, Feb., 1954.

In retinal elastosis, a hemeralopia of the posterior pole can be demonstrated by scotopic campimetry and occasionally confirmed by a subnormal electroretinographic response. When it is considered that angioid streaks are often associated with choroidal sclerosis and with macular degeneration of the senile exudative type these findings suggest that the condition is in reality a tapetoretinal degeneration at the posterior pole. (21 figures, 240 references) John C. Locke.

Henkes, Harold E. **Electroretinography in circulatory disturbances of the retina. II. The electroretinogram in cases of occlusion of the central retinal artery or of one of its branches.** A.M.A. Arch. Ophth. **51**:42-53, Jan., 1954.

This article reports on 31 cases of occlusion of the central retinal artery or of one of the retinal arteries and discusses vasodilatory therapy from an electroretinographic standpoint. A negative − electroretinogram is typical of central artery occlusion. A negative + suggests a branch occlusion. The success of vasodilator therapy depends upon early institution of treatment. The most potent vasodilators are tolazoline hydrochloride 0.020 gm. and Roniacol tartrate 0.1 gm. given intravenously. In seven of eleven eyes in which treatment was started within the first four days, there was improvement in visual acuity. In older cases only one in seven showed improvement. A close correlation between improved visual acuity and ret-

inographic changes could not be demonstrated. (8 figures, 1 table, 5 references)
G. S. Tyner.

Henkes, Harold E. **Electroretinography in circulatory disturbances of the retina. III. Electroretinogram in cases of senile degeneration of the macular area.** A.M.A. Arch. Ophth. **51**:54-66, Jan., 1954.

Eighty cases of senile macular degeneration were studied by electrographic methods to demonstrate, if possible, alterations in retinal or choroidal circulation. In general, the electroretinogram showed widespread circulatory impairment in the choriocapillaris. Vasodilator therapy was not a successful method of treatment. (10 figures, 1 table, 7 references) G. S. Tyner.

Ingalls, T. H., and Purshottam, N. **Oxygenation and retrolental fibroplasia.** New England J. Med. **250**:621-629, April 15, 1954.

The authors have briefly reviewed the literature on this subject from the epidemiological point of view. They stress the importance of histotoxic anoxia as brought about by thermodynamic derangements, and subscribe to the hypothesis of optimum oxygen level maintenance. (1 color plate, 4 figures, 2 tables, 65 references) H. Horwich.

Kronfeld, P. C., and Pischel, D. K. **Scleral resection operation for retinal detachment.** A.M.A. Arch. Ophth. **51**:356-363, March, 1954.

The authors report their results in two hundred consecutive scleral resection operations which they had performed in cases of idiopathic and post-traumatic retinal detachment. The study was undertaken 1. to learn more about the processes operative in retinal detachments; 2. to define more accurately the indications for the scleral resection operation, and 3. to define more accurately the mode of action of the scleral resection operation. The predominant factors in the development of detachment were considered to be shortness of the retina, star folds, and traction from within. The operation proved successful primarily in cases of shortness of retina, but also for star folds and traction from within. The beneficial effect of the operation was probably due to the creation of a state of relative slackness of the retina and of elements of traction within the vitreous. In their series 62 operations were successful, 17 eyes were improved and in 121 there was failure. (2 tables, 11 references) R. W. Danielson.

Lavel, J., and Collier, R. **Spontaneous hole in anterior hyaloid membrane.** A.M.A. Arch. Ophth. **51**:67-68, Jan., 1954.

Ten weeks after intracapsular cataract extraction a small hole was seen in the anterior hyaloid somewhat temporal and below the center. Strands of vitreous protruded from the hole into the anterior chamber. No visual disturbance was noted. (1 figure, 2 references)
G. S. Tyner.

Locke, John C. **Retrolental fibroplasia.** A.M.A. Arch. Ophth. **51**:73-79, Jan., 1954.

Studies are reported which support the hyperoxygen theory of the etiology of retrolental fibroplasia. The incidence of retrolental fibroplasia was less in children who received minimal amounts of oxygen and higher in those who received higher concentrations of oxygen. No conclusions were possible regarding the effect of rapid vs. gradual withdrawal of oxygen. (4 tables, 14 references) G. S. Tyner.

Massin, Marcel. **Traumatic detachment of the retina.** Arch. d'opht. **14**:154-171, 1954.

The author notes the important role of traumatism in the production of retinal detachment and its industrial significance in relation to compensation insurance. In

a review of the literature and from a study of 22 personal cases, he has attempted to define this role, particularly in regard to the lapse of time between the injury and the development of the detachment. He has reached the following conclusions. 1. A contusion in a healthy eye does not usually lead to immediate detachment but may lead to an atrophic or proliferating retinopathy from which detachment arises after a variable time interval. 2. A penetrating injury does not provoke immediate detachment but is usually followed by a massive cicatricial chorioretinitis which fixes the retina to the choroid. Later a detachment may occur. 3. Retinal detachment, after post-traumatic cicatrizing chorioretinitis, is nevertheless of infrequent occurrence after either contusion or penetrating injury. 4. A special type of detachment in young subjects, i.e., a flat, inferior separation with disinsertion in the inferotemporal quadrant, is post-traumatic in about one-third of the cases and is due primarily to a peripheral chorioretinitis. 5. Traumatism, direct or indirect, can play a role, often difficult to define, in the origin of a senile or simple myopic detachment. (30 references) P. Thygeson.

Patz, Arnell. **Clinical and experimental studies on the role of oxygen in retrolental fibroplasia.** Tr. Am. Acad. Ophth. **58**:45-50, Jan.-Feb., 1954.

The ocular and systemic effects of oxygen administration on several species of animals are presented. Where newborn or young mice, rats, kittens, and dogs were exposed to increased oxygen environment, the characteristic lesions of human retrolental fibroplasia were produced. These consisted of nodules of endothelial cell proliferation in the retina, budding of capillaries from the retina into the vitreous, retinal hemorrhages, and retinal edema. The lesions were proportional in severity to the duration and concentration of oxygen administered. The results of a controlled nursery study which implicate prolonged high oxygen administration as an important factor in the development of retrolental fibroplasia are cited. These data merit further clinical and experimental study to clarify the mechanism of oxygen action and to explore the role of other factors probably associated with oxygen administration. The data thus far collected justify the recommendation that oxygen therapy be curtailed in the premature nursery to limits compatible with the physical status of the premature infant. (4 figures, 10 references) Theodore M. Shapira.

Pensani, Bruno. **Meteorology and retinal detachment. The influence of some meteorological and climatic factors on the frequency of the condition.** Ann. di otttal. e clin. ocul. **79**:23-32, Jan., 1953.

Correlating meteorological data with the development of 130 cases of detachment of the retina over a period of seven years, suggested that the atmospheric vapor-tension influences the appearance of retinal detachment. Temperature and barometric pressure are of no, or very minor, importance. Sixty-two percent of detachments began in autumn or winter, 38 percent in spring or summer. (3 tables, 11 references) John J. Stern.

Rais, Ben Slimane, and Mrad. **A case of lateroversion of the retina.** Arch. d'opht. **13**:773-775, 1953.

A case of lateroversion of the retina occurring in a 32-year-old native Tunisian is presented and illustrated by a fundus drawing in black and white. An enormous tear extending from 7 to 12 o'clock, rather than the usual disinsertion, was responsible for the condition. There was no history of traumatism but there was a myopia of minus 18 prism diopters. The retinal flap, instead of being turned back

completely over the disc, was seen to be floating anteriorly in the vitreous, contrary to the reported findings in inferoversion. Also unlike inferoversion, the vessels could not be seen by transillumination on the posterior surface of the retina. The authors conclude that no surgical or medical treatment is yet available for this lesion. (1 figure) P. Thygeson.

Stagni, Severino. **The relation between the insertion of the extrinsic muscles of the eye and the site of retinal tears.** Ann. di ottal. e clin. ocul. 79:41-53. Jan., 1954.

In 51 of 72 cases of detachment the retinal tear occurred near the insertion of one of the extrinsic muscles. In the remaining 21 cases, where no relation was found, there was aphakia, cystic detachment, extensive retinal degeneration, or more than one tear. John J. Stern.

Straatsma, Bradley R. **Angiomatosis retinae.** New England M. J. 250:314-317, Feb., 25, 1954.

A case of angiomatosis retinae is reported wherein the author used diathermy coagulation after he had placed a Walker pin to mark the area occupied by the tumor mass. Vision remained at 20/20 in the affected eye. (2 figures, 32 references) Irwin E. Gaynon.

Strazzi, Athos. **Retinitis pigmentosa and endocrine disturbances.** Riv. otoneuro-oftal. 29:145-169, March-April, 1954.

The author, after reviewing briefly the syndromes of Bardet-Biedl and of Lawrence-Moon and the several retinopathies related to retinitis pigmentosa, presents very detailed case histories of two brothers in whom, at the ages of seven and 13 , a diagnosis of Bardet-Biedl syndrome with retinitis pigmentosa sine pigmento was made, but in the older brother a typical retinitis pigmentosa was diagnosed when he reached 18 years of age. The author suggests that a retinitis pigmentosa sine pigmento should be considered as the first stage of a typical pigmentosa and one of the tapeto-retinal degenerations.

A third case history is that of a patient with hypogenitalism on a diencephalic-hypophyseal basis, who had hemeralopia and an absolutely normal fundus picture. After several years he developed scotomata and migration of pigment.

The author assumes that the retinitis pigmentosa and the diencephalic symptoms are due to the same cause in the first two patients, but in the third, direct correlation between the retinal and the diencephalic signs is not obvious unless the diencephalic disturbance is considered necessary for the development of the retinal abnormalities, be they primary or the result of a secondary arachnoencephalitis. (7 figures, 17 references)

Walter Mayer.

12

OPTIC NERVE AND CHIASM

Huerkamp, B. **Ophthalmoscopic findings in Leber's disease.** Klin. Monatsbl. f. Augenh. 124:164-170, 1954.

A family tree is presented through four generations. Primary optic atrophy occurred in five members of three generations of this family. In addition to the changes of the optic disc, retinal edema, hemorrhages, pigmentation, and sheathing of vessels could be observed in the fundi of these patients. One of them had a ring scotoma. A meningeal involvement in this disease is considered. (1 figure, 23 references) Frederick C. Blodi.

Siegert, P. **Remarks on Kreibig's paper on opticomalacia.** Klin. Monatsbl. f. Augenh. 124:337-338, 1954.

The paper of Kreibig (Klin. Monatsbl. f. Augenh. 122:719) describes opticomalacia as a sequel of vascular obstruction in

the retrobulbar part of the optic nerve. This occurred together with temporal arteritis and is one manifestation of it. Siegert believes that ischemia of the disc also occurs with temporal arteritis.

Frederick C. Blodi.

13
NEURO-OPHTHALMOLOGY

Cogan, David C. **Ocular dysmetria; flutter-like oscillations of the eyes, and opsoclonus.** A.M.A. Arch. Ophth. **51**:318-335, March, 1954.

The phenomena here designated ocular motor dysmetria, flutter-like oscillations, and opsoclonus have rarely been refered to in the literature. Ocular dysmetria may be said to be preesnt when a person with normal visual fields and normal visual acuity shows consistently an overshoot and lack of precision in changes of fixation. Cogan reports six cases, in all of which there was evidence of a disturbance in the cerebellum or cerebellar pathways. Eleven patients with flutter-like oscillations are described. The phenomenon is characterized by periodic movements in the horizontal plane, lasting no more than a few seconds. These oscillations are often spontaneous, but more frequently are precipitated by changes of fixation. They differ from dysmetria in being cycles of approximately equal amplitude and are also associated with cerebellar disease. Two cases of opsoclonus are presented. This movement differs from "flutter" in being more sustained, totally irregular, and occurring in the vertical, as well as the horizontal plane. There is no substantial basis for believing that these are cerebellar in origin. (2 charts, 20 references)

R. W. Danielson.

Ishigôoke, K. **Ocular symptoms due to electric stimulation of the superior colliculus.** Acta Soc. Ophth. Japan **58**:135-154, & 191-201, Feb.-March., 1954.

A stimulation of the superficial layer of the superior colliculus in the cat results in a miosis of the opposite side with closure of the lids. Stimulation of the third layer of the colliculus, the substantia grisea centralis, and the formatio reticularis, results in mydriasis with opening of the lids. The stimulation of the colliculus also causes horizontal movements of the eye towards the opposite side, whereas a stimulation of the fasciculus longitudinalis posterior causes movements towards the corresponding side. The author believes that there is a crossing of the fasciculus fibers. A stimulation of the commisura colliculorum superior causes convergence. A pure vertical movement of the eye (a subcortical center has been assumed to be in the superior colliculus) is not brought about by a stimulation of the colliculus. (13 figures, 102 references)

Yukihiko Mitsui.

Pignalosa, Giuseppe. **A case of crocodile tears.** Arch. di ottal. **58**:75-83, Jan.-Feb., 1954.

Pignalosa describes a patient in whom the syndrome of crocodile tears was associated with a sensation of noise in the ipsolateral ear during mastication. He suggests that the ear disturbance is caused by a concomitant lesion of the stapedius and that the lesion of the facial nerve is situated at the level of the geniculate ganglion. (16 references)

John J. Stern.

Stokes, E. H. **Thrombosis of the left posterior inferior cerebellar artery.** M.J. Australia. **1**:450, March 20, 1954.

The ocular signs included left miosis, left lateral rectus palsy and nystagmus. Symptoms were pain of sudden onset in the left side of the face and precordium, hoarseness, dysphagia and giddiness. These are explained on an anatomical basis according to the region of the brain stem which was involved.

Ronald Lowe.

Venturi, G., and Pacifico, G. **Ophthalmologic findings in electronarcosis.** Boll. d'ocul. **33**:40-51, Jan., 1954.

Thirty otherwise sound patients with schizophrenia or paranoia, aged between 20 and 55 years, were examined before, during and after electroshock, and one or two days later. Lid reflexes, extraocular muscles, cornea, conjunctiva, pupils, eye-pressure, the refractile media, the retinal arterial pressure, and the fundi were examined. During the tonic phase, contraction of the orbicularis oculi, profuse lacrimation, miosis, absence of pupillary light reaction, eye pressure increase, increased retinal arterial pressure, and hyperemia of the optic nerveheads were found; during the narcotic phase, conjugate deviation, compensatory eye movements following sudden passive head movements, corneal and conjunctival anesthesia for four to five minutes, and conjunctival hyperemia lasting some hours after treatment were manifest; pupillary light reflexes were present, and in some of the patients subnormal eye pressure. A return to normal values occurred one half to two hours later. Seldom were angiospasm or venous stasis found. The findings are compared with ocular findings in insulin shock, cardiazol shock and acetylcholin shock. (30 references)

K. W. Ascher.

Vianna, E. Velloso. **Herpes zoster ophthalmicus.** Arq. brasil. de oftal. **16**:177-180, 1953.

Herpes zoster ophthalmicus in an eight-year-old child was treated by radiation, 220 r. were administered twice weekly. The author was unable to find any previous report of the use of radiation therapy in this particular type of zoster, and presents this case to demonstrate the unusual features and success of therapy. Pain ceased the day after the first treatment, and the lesions gradually regressed. Photophobia did not subside as readily, however. (2 figures)

James W. Brennan.

14

EYEBALL, ORBIT, SINUSES

Bignell, John L. **The "blind and the painful" eye.** M. J. Australia **11**:667-669, May 1, 1954.

Emphasis is placed on the importance of retaining a blind eye whenever possible for both psychologic and cosmetic reasons. Various procedures are discussed including cyclodiathermy and alcohol injection. Methods to assist accurate diagnosis are briefly mentioned. Ronald Lowe.

Bonheure. **A case of intraobital tumor.** Ann. d'ocul. **187**:178-183, Feb., 1954.

This report of a case of subperiosteal dermoid cyst of the orbit with atypical clinical manifestations illustrates the difficulties in the diagnosis of many orbital tumors by methods other than surgical intervention. (2 figures) John C. Locke.

Dollar, J. M., and Savory, M. **Exenteration of the orbit and use of alginate mould for applying skin grafts.** Brit. J. Ophth. **38**:39-45, Jan. 1954.

Exenteration of the orbit is a very unsatisfactory procedure but often there is no other choice and it must be done. Diathermy has made the operation easier and now the after-care of the orbit is the principal concern. Since the procedure leaves bare bone exposed, it is better to withhold skin grafting until the bone is covered by a layer of granulation tissue, which comes in from the skin margins; this ordinarily takes two weeks but helps to insure successful take of the graft. Success depends in a large measure on the graft being maintained by firm, even pressure on all of its surface. This is easily done by the use of an alginate mold made of sodium alginate, which sets to a

jelly on the addition of calcium chloride solution, mixed with pieces of cotton to add firmness.

The technique of the exenteration is described. The incision is made through the skin of the lids just inside the orbital margins; the periosteum must be completely and carefully removed with the rest of the orbital contents. The mold is made and allowed to set in the cavity while the graft is being removed from the thigh, then removed and put back into the orbit over the graft, which is not sutured. It is removed after a week, and vaseline dressings are applied. As soon as the cavity is healed it is fitted with a prosthesis of which there are two types; one fits onto a spectacle and the other is attached to the skin by an adhesive tape. (5 figures) Morris Kaplan.

Faldi, S. **Further clinical trials with Copper's orbitonometer.** Gior. ital. oftal. **4**:498-502, Sept.-Oct., 1953.

The value of this instrument in the various conditions giving rise to exophthalmos is emphasized, and graphic results in a case of each of the following lesions are reported: orbital aneurysm, pulsating exophthalmos, angiofibroma of the orbit, orbital cellulitis, cholesteatoma of the orbit, and sarcoma of the orbit. There is a striking difference between the results in exophthalmos due to vascular conditions and to neoplastic causes. V. Tabone.

Illig, K. M. **A secure fixation of the implant after enucleation.** Klin. Monatsbl. f. Augenh. **124**:200-201, 1954.

The anterior sclera with the muscle insertions remains in the orbit. An evisceration is performed with excision of the posterior sclera. The plastic ball is inserted back of the remaining scleral ring. Frederick C. Blodi.

Manchester, P. Thomas. **The X-ray diagnosis of orbital tumors.** South. M. J. **47**:231-234, March, 1954.

An X-ray diagnosis of tumor of the orbit may be made by 1. tomography wherein various depths of the orbit are studied, 2. injection of air into the orbit, 3. cerebral arteriography with the increased staining produced by vascular tumors of the orbit, and 4. orbitography wherein radioopaque dyes are injected directly into the orbit. As the liquid fills the orbit, it spreads about the tumor and outlines it. (5 references) Irwin E. Gaynon.

Pajtas, Jozef. **Ten-year experience with an amber orbital implant wrapped in the patient's fat tissue.** Bratislavske Lekarske Listy **33**:945-950, Oct., 1953.

In the period 1939-1952 the author implanted used natural or artificial amber wrapped in the patient's fat tissue into the orbit in 163 cases. Twelve were extruded soon after the operation and three more were lost after two years. The authors believe that this type of combined implant is well tolerated by the tissues. It gives satisfactory, well-healed implants in 90 percent of cases. The motility of the implant is good though good movement was transmitted to a prosthesis only in 50 percent of cases. Buried implants are still more practical than the integrated implants because of more frequent extrusion of the latter. (13 references) Sylvan Brandon.

Pearlman, Maurice D. **Recommendations for the utilization of polyvinyl plastic sponge as an orbital implant.** Tr. Am. Acad. Ophth. **57**:910-911, Nov.-Dec., 1953.

The author states that this surgical technique has effected satisfactory cosmetic results in a series of 60 patients observed as long as 24 months. About 9 percent of the cases were unsuccessful because of irreparable exposures of the implant face and consequent infection which developed during the early postoperative weeks. In the remaining 91 percent, the pa-

tients have been free from exposures and extrusions for as long as 24 months. This early complication may be obviated by attaching the muscles onto the anterior face of the implant, and such a series is now under development. (1 figure)

Theodore M. Shapira.

Stokes, E. H. **Juvenile thyrotoxicosis.** M. J. Australia **1**:450, March 20, 1954.

Thyrotoxicosis with severe exophthalmos in a girl, aged 16 years, is described.

Ronald Lowe.

15

EYELIDS, LACRIMAL APPARATUS

Braley, Alson E. **The lids, lacrimal apparatus, and conjunctiva.** A.M.A. Arch. Ophth. **51**:91-137, Jan., 1954.

The literature from the summer of 1952 to the summer of 1953 is reviewed. Many valuable hints are given as to etiology and treatment of common disorders seen in the average office practice. (1 table, 321 references) G. S. Tyner.

Clark, James W. **Obstructions of the lacrimal duct.** Illinois M. J. **105**:183-185, April, 1954.

A trocar with an obturator shaped like a Ziegler probe is passed through the natural bony opening after dilatation of the puncta and canaliculi with Ziegler probes. The obturator is then removed and a plastic tube is inserted and so placed that the upper end of the tube remains in the superior portion of the lacrimal sac. The inferior portion of the tube is allowed to remain free in the nose.

Irwin E. Gaynon.

Keiden, S. E. **Elephantiasis nostras.** Lancet **1**:79-81, Jan. 9, 1954.

Persistent edema of the eyelids following a traumatic hematoma of the forehead and subsequent recurrent attacks of erysipelas in an eight-year-old child are described. (2 figures) Irwin E. Gaynon.

Kleberger, E. **Our experiences with the reconstruction of a lacrimal duct using supramid tubes.** Klin. Monatsbl. f. Augenh. **124**:290-296, 1954.

The immediate results were excellent and epiphora disappeared in 20 patients who were operated on. However, in all but four of these patients late complications and stenosis occurred. These poor final results confirm the impressions of Papst (Klin. Monatsbl. f. Augenh. 123:58, 1953) and induced the author to return to dacryocystorhinostomy. (3 figures, 7 references) Frederick C. Blodi.

de Saint-Martin, R. **Plastic repair with a sliding flap. Treatment of choice for the delayed repair of evulsion of the lower lid.** Ann. d'ocul. **187**:209-226, March, 1954.

The author describes a method of using a sliding flap in the late repair of an old evulsion of the lower lid, where there has been injury to the inner extremity of the lid margin. (12 figures) John C. Locke.

Young, Robert J. **Congenital ectropion of the upper lids.** Arch. Dis. Childhood **29**:97-100, April, 1954.

A case of congenital ectropion of the upper lids in a mongolian idiot is reported. The constricting effect of the marked over-development of the epicanthic fold which pressed on the outer surface of each upper lid in the region of the inner canthus hindered the venous blood flow. As a result the conjunctiva became sufficiently swollen to evert the lid. (1 figure, 1 table, 6 references) Irwin E. Gaynon.

16

TUMORS

Cook, Charles. **Uveal lymphosarcoma.** Brit. J. Ophth. **38**:182-185, March, 1954.

Lymphomatous tumors of the adnexa and orbit have been found rather frequently but similar intraocular tumors have been seen very rarely. In the London

Institute of Ophthalmology during the past ten years, only one intraocular tumor has been found in all the biopsies of lymphomatous tumors. A 59-year-old woman had sudden loss of vision two years before, with no other symptoms except sudden, severe, painful glaucoma at the time of enucleation for what was thought to be thrombotic glaucoma. A gelatinous mass was found in the orbit adherent to the globe and was found to be lymphosarcoma primary in the choroid, where it had reached massive proportions. The patient recovered from the operation and then received a course of deep X-ray therapy to the socket. In two and a half years there has been no evidence of recurrence. This tumor is characteristically radiosensitive so that surgical removal with X-ray therapy may offer a relatively good prognosis. (3 figures, 8 references)

Morris Kaplan.

Faldi, S. **Cavernous angioma of the conjunctiva, in a case with Recklinghausen's disease.** Gior. ital. oftal. **4**:465-488, Sept.-Oct., 1953.

A case is described in which the appearance of a cavernous angioma of the conjunctiva coincided with an exacerbation and evolution of neurofibromatosis of the lid of the same side. The lid condition had been quiescent for ten years. The possible causes of Recklinghausen's disease, as well as its relations to the other phacomatoses, are discussed at length. The author suggests that all these allied developmental aberrations could be grouped together under one vague and indefinite heading. V. Tabone.

François, J., Rabaey, M., and Debeir, O. **Eosinophilic granuloma of the orbit.** Ann. d'ocul. **187**:165-177, Feb., 1954.

The authors report the second known case of eosinophilic granuloma of the orbit without lesions in the bones. Clinical manifestations which suggest this diagnosis are a swelling of the upper lid in a child or young adult, the demonstration by Xray of a well-defined osteolytic process without reactive bone formation at its margin, and the presence of a blood and medullary eosinophilia. The diagnosis is confirmed by biopsy. The cause is unknown, the prognosis good. Surgical excision is effective, but unnecessary, since radiotherapy gives good results. (8 figures, 24 references) John C. Locke.

Grosse, Hans. **Melanoma and melanosis conjunctivae.** Arch. f. Ophth. **154**:368-380, 1953.

Six epibulbar melanomas were anatomically studied. Pigment granules were either found within the epithelial cells or in melanoma cells situated between the epithelial tissue. The possibility is considered that melanomas originate from the ectodermal as well as the mesodermal layer of the embryo. (4 figures, 1 table, 144 references) Ernst Schmerl.

Györffy, St. **Epibulbar tumor of unusual size.** Klin. Monatsbl. f. Augenh. **124**:339-341, 1954.

A 67-year-old farmer came to the clinic with a tumor on the right eye. This tumor projected 60 mm. in front of the corneal plane and was up to 45 mm. wide. Histologic examination after exenteration revealed an epidermoid tumor, probably stemming from the limbus. (2 figures)

Frederick C. Blodi.

Samuels, Bernard. **Melanosis oculi and sector-shaped melanosis and melanosarcoma of the choroid.** Arch. f. Ophth. **154**: 361-367, 1953.

One case of melanosis and melanosarcoma was studied histologically. The melanosarcoma seemed to have developed independently from the pigmented area of the choroid. (6 figures, 15 references)

Ernst Schmerl.

17
INJURIES

Chrzanowska, Krystyna. **Ocular trauma in adults.** Klinika Oczna **23**:277-284, 1953.

Injury to the eyes were present in 20 percent of the patients over 16 years of age in the Krakow Eye Clinic in 1951 and 1952. Slight injuries were seen in 90 percent, mostly foreign bodies of the cornea in men working on machines. Only 8 percent of the total of slight injuries occurred in women. 10 percent of the injuries were severe. Not all of the injuries were ascribed to work, but to other accidents, injuries at home, fights, and explosives. Among the workers those working in metal industry were in majority. In women farm work caused the greatest number of injuries. Blindness resulted in 22 percent of severe injuries, visual loss to 6/12 was seen in 43 percent. Magnetic foreign bodies were removed in 70 percent and vision was saved in 63 percent of cases. Improvement is noticed by comparison with the report of Prof. Wilczek for the years 1938 to 1939. Use of proper safety glasses would have decreased the number of severe injuries by 8.4 percent and slight injuries by 20.3 percent. (3 tables) S. Brandon.

Domaszewicz, Wanda. **Burns of the eyes and their treatment.** Klinika Oczna **23**:285-288, 1953.

The importance of an ophthalmologist in treatment and prevention of loss of vision in eye burns is emphasized. Caustic burns are the most frequent, lime being the cause in most cases. Acid and thermal burns are less frequent. Appearance, treatment and the outcome of a severe burn is described. The author uses 1-percent solution of tannic acid for irrigation the first two days. Later, saline solution is used. Antibiotic ointment for prevention of infection and mydriatics are used from the beginning. To prevent adhesion in the fornices massage with a glass rod is advocated. Paracentesis of the anterior chamber is advised in severe lime burns. Clearing of the cornea was observed after subconjunctival injection of aqueous extracts of placenta. Three cases of severe burns in young boys are presented for illustration. First aid immediately after the accident is of utmost importance. The chemical should be washed out of the conjunctival sac and the fornices.

S. Brandon.

Flynn, G., and Raiford, M. **Beryllium and delayed corneal healing.** A.M.A. Arch. Ophth: **51**:89-90, Jan., 1954.

A case is reported in which delayed corneal healing occurred after removal of a corneal foreign body (glass from a fluorescent bulb). The authors apparently assumed the delay in healing to be due to beryllium contamination at the time of injury. G. S. Tyner.

Madroszkiewicz, Marian. **New case of severe traumatic cyclitis is cured by a contrasting suture around the limbus.** Klinika Oczna **23**:289-291, 1953.

Severe traumatic cyclitis in a man, 47 years of age, is described. An intraocular nonmagnetic foreign body was present which could not be removed. Usual conservative treatment was unsuccessful. The author put a catgut suture impregnated with barium salts around the limbus for 24 hours. Within a few hours the eye became quiet and started to respond to mydriatics and other drugs. The final vision was 6/24. The foreign body was still present but the eye was perfectly quiet. (3 references). S. Brandon.

Schofield, A. L. **A review of burns of the eyelids and their treatment.** Brit. J. Plast. Surg. **7**:67-91, April, 1954.

The author deals with flash, gasoline, fire, acid, and phosphorus bomb burns of the eyelids and the treatment of the re-

sultant ectropion by the epithelial outlay graft technique. (21 figures, 2 references)
Irwin E. Gaynon.

18

SYSTEMIC DISEASE AND PARASITES

Arouh, J., Zambrano, J., and Lis, M. **Ocular manifestation in botulism.** Rev. oto-neuro-oftal. Sudam. **29**:19-25, Jan.-Feb., 1954.

The authors summarize the generalities of botulism and the characteristics of the Clostridium botulinum. The most common eye manifestations are paralysis of external ocular muscles, ptosis, mydriasis, and cycloplegia. There may also be a nystagmus and hyper- or hyposecretion of tears. In certain cases an optic neuritis has been described. The authors give the findings in two patients with botulism, one of whom died a few days after admission to the hospital. The other recovered after three months. The authors attribute a great prognostic importance to the possible recurrence of pupillary disturbances which always precede, for a few hours, a generalized recurrence of the disease.
Walter Mayer.

Bonnet, Paul. **Temporal arteritis and its ocular manifestations. A critical review.** Arch. d'opht. **14**:24-47, 1954.

The author describes temporal arteritis as a disease of the aged, characterized essentially by a cephalalgia of temporal localization, often intolerable. The disease, in which the temporal artery is indurated and painful to palpation, consists actually of a generalized arteritis which is histologically a granulomatous inflammation with giant cells. A sudden loss of vision, first in one eye and then in the other, may occur at any time during the evolution of the disease, and unilateral or bilateral blindness occurs in about 25 percent of patients. Treatment, which has included the use of cortisone and ACTH, has been unavailing. The author notes that the sedimentation rate remains high during the entire clinical course of the disease. The literature is adequately reviewed. (78 references) P. Thygeson.

Ditzel, Jörn. **Morphologic and hemodynamic changes in the smaller blood vessels in diabetes mellitus. I. Considerations based on the literature.** New England J. Med. **250**:541-546, April 1, 1954.

In reviewing the recent literature pertinent to this subject, it appears that the capillary endothelium and basement membrane are the most important tissues. The modern hypotheses for the changes occurring there suggest that there is an impairment of the blood circulation in the terminal peripheral vascular system leading to prolonged suboxidation and subnutrition of the endothelium. A metabolic disorder of the simple polysaccharides (glycogen) or complex polysaccharides (mucopolysaccharides) involves the basement membranes of the capillaries and the ground substance of connective tissue. (76 references) H. Horwich.

Ditzel, J., and Sagild, U. **Morphologic and hemodynamic changes in the smaller blood vessels in diabetes mellitus. II. The degenerative and hemodynamic changes in the bulbar conjunctiva of normotensive diabetic patients.** New England J. Med. **250**:587-594, April 8, 1954.

One hundred and fifty diabetics and 90 non-diabetics, free from complications such as hypertension or infection, were studied by the stereoscopic microscope method of Knisely. They were divided into groups according to age, sex, and severity of disease. Both vascular and extravascular changes were evaluated. The most consistent findings in the diabetic subjects were longation of the venous part of the capillaries, distention of the venules, and aggregation of the blood cells, with concomitant reduction of blood flow. The authors conclude that certain primary changes in the membrane

function of the capillaries take place in the diabetic condition, and suggest that changes in the serum proteins may be of basic importance. (6 figures, 23 references) H. Horwich.

Ehrlich, J. C., and Greenberg, D. **Sicca syndrome.** A.M.A. Arch. Internal Med. **93**:731-741, May, 1954.

A case of Sjögren's syndrome in a 49-year-old white woman is reported. Clinical manifestations included keratoconjunctivitis sicca, dryness of the mucous membranes, bilateral enlargement of the parotid glands, with absence of secreting glands shown by biopsy, and rheumatoid arthritis. (5 figures, 45 references)

Irwin E. Gaynon.

Heath, Christopher. **A case of meningitis apparently secondary to herpetic keratitis and possibly precipitated by cortisone treatment.** Brit. J. Ophth. **38**:58-60, Jan., 1954.

A 43-year-old man had repeated attacks of dendritic ulcer in the right eye for several years, with no other complications. During one attack a generalized keratitis with uveitis developed and after atropine sensitivity appeared, a ½ percent solution of cortisone was instilled twice daily. After three days the patient became violently ill with acute meningitis; no causative agent was found in the turbid spinal fluid. He responded promptly to aureomycin therapy and eventually the eye healed also. The meningitis was ascribed to the virus of herpes simplex, the virulence of which became manifest after the cortisone had reduced the local resistance to the infection.

Morris Kaplan.

Kiel, Eduard. **Ophthalmomyiasis.** Klin. Monatsbl. f. Augenh. **124**:194-200, 1954.

Three cases are mentioned, two of which were reported previously (Kl. Mbl. Augenh. 88:835, and Kl. Mbl. Augenh. 78, 1927). In the first patient the larva of the fly was under the upper lid, in the second the larva was in the anterior chamber and could be removed. The third patient, a 9-year-old boy (practically all these patients are children), had a larva in the anterior chamber which migrated into the vitreous. The eye was eventually lost. The author believes that in all cases of internal myiasis the larva of the fly penetrates the sclera and first reaches the vitreous. From there it may migrate into the anterior chamber. (11 references)

Frederick C. Blodi.

Leibiger, W. **Ocular involvement in endangiitis obliterans.** Klin. Monatsbl. f. Augenh. **124**:334-337, 1954.

A 55-year-old man with endangiitis obliterans showed sludging in the conjunctival and retinal blood vessels. Newformed blood vessels could be seen on the iris and on the disc. Cataract occurred suddenly in one eye. (2 figures)

Frederick C. Blodi.

Nastri, F., and D'Ermo, F. **Protein content of the blood of diabetics with retinopathy.** Boll. d'ocul. **33**:3-10, Jan., 1954.

In diabetics with retinopathy, the reduction of blood proteins and the increase in beta and gamma globulin is more marked if the disease is complicated by renal and hepatic affection. The chemical alterations may predispose to the ocular lesions. (5 tables, 14 references)

K. W. Ascher.

Pagliarani, N. **Ocular findings in children born of mothers after toxemia of pregnancy.** Boll. d'ocul. **32**:705-713, Dec., 1953.

The eyes of 103 newborn infants of mothers who had had toxemia of pregnancy, and children aged one to six years who were born in the course of a severe maternal eclampsia were examined ophthalmoscopically. The eyes of two deceased newborn infants of eclamptic mothers were studied histologically. High

incidence of ocular and cerebral hemorrhage was found. The latter may lead to ophthalmoplegias and optic nerve atrophy. The retinal hemorrhages disappear, often without permanent damage. (29 references) K. W. Ascher.

Piper, H. F. **Astigmatism, watchglass nails and bronchiectasis as a systemic disease.** Klin. Monatsbl. f. Augenh. **124**:188-194, 1954.

The author believes that there is a frequent association between these three conditions. Six cases of increasing astigmatism (one with keratoconus) in young adults are cited. (1 figure, 10 references) Frederick C. Blodi.

Strazzi, Athos. **Myasthenia gravis.** Riv. oto-neuro-oftal. **29**:170-181, March-April, 1954.

The author reviews the history of myasthenia gravis and emphasizes the fact that the pathogenesis of myasthenia gravis is still unknown. He suggests that the essential defect lies in the humoral transmission at the myoneural junction and is probably the result of a complex endocrine dysfunction. He describes the case history of a woman with signs of hyperthyroidism, suprarenal deficiency, and an increase in creatinuria and blood cholinesterase. This patient also had myasthenia, of which the only ocular signs were ptosis and weakness of the oculomotor muscles for over a year. The last of the symptoms was revealed only after the fatigue test. (13 references) Walter Mayer.

Thomas, C., Cordier, J., Algan, B., and Fiel, J. **Visual accidents in the course of intervention on the pleura.** Arch. d'opht. **14**:5-23, 1954.

The authors note that visual and other neurological accidents have been known since 1850 to occur in connection with pulmonary collapse therapy for tuberculosis and other procedures involving the pleura. The visual accidents have sometimes accompanied other central nervous symptoms of a convulsive or paralytic nature. In the first of their two cases an intrapleural injection of lipiodol for visualization of a cavity resulted in a transient left hemi-paralysis, a coma of 30 minutes duration, and a subsequent amaurosis relieved by vasodilators. In their second case, air injection to collapse a tuberculous lung led to sudden reduction of vision to 1/50 O.U. and to a left hemiparalysis. Visual field study indicated a double hemianopsia with conservation of macular vision only. Therapy with vasodilators led to a return of visual acuity to 20/20 and to a normal visual field within three days.

The theories of the mechanism of the production of these accidents are discussed in detail. The authors consider the embolus theory more likely than the pleural reflex theory. They consider vasodilator therapy essential for relief of the arterial spasm provoked by the embolus. (96 references) P. Thygeson.

Vanni, V. and Frugoni, P. **Rare ocular manifestations of Raynaud's disease.** Boll. d'ocul. **32**:741-764, Dec., 1953.

In the literature, retinal arteriospasm, venous thrombosis, arterial occlusion and papilledema have been described as complications of Raynaud's syndrome. The authors describe a woman, aged 46 years, who had headache, papilledema in one eye, and secondary optic nerve atrophy in the other; the lesions caused defective vision and asymmetric visual field defects, which, in the right eye, were mainly in the nasal lower quadrant, and in the left eye in upper nasal and part of the upper temporal quadrants. Vasospasms were assumed to be responsible. A bilateral cervical sympathectomy was performed and was followed by remarkable improvement of function in both eyes. The figures exhibit a photograph of the patients, visual

fields before and after surgery, drawing of the disc in color, and eight X-ray photographs of the skull taken during arteriography and after air injection. (12 figures, 34 references) K. W. Ascher.

Viallefont, H. **The oculosalivary syndromes.** Ann. d'ocul. **187**:145-156, Feb., 1954.

Several syndromes in which the lacrimal and salivary glands are both affected are described, e.g.: Stevens-Johnson's syndrome, Behçet's syndrome, mumps, Heerfordt's syndrome, and Gougerot-Sjögren's syndrome. John C. Locke.

19

CONGENITAL DEFORMITIES, HEREDITY

Falls, Harold F. **Clinical detection of the genetic carrier state in ophthalmic pathology.** Tr. Am. Acad. Ophth. **57**:858-872, Nov.-Dec., 1953.

The carrier states of genetically determined ocular abnormalities are discussed, utilizing the classification developed by Neel and Schull. Throughout, it is emphasized that a high index of suspicion or awareness of the hereditary nature of the various ocular and systemic entities constitutes a prime requisite to detection of the carrier states in genetic ocular pathology. Many clinically, apparently similar, genetic ocular entities have different gene transmission patterns (dominant, recessive and sex-linked modes of inheritance exist). This phenomenon makes it imperative that the individual as well as his family be carefully studied before giving genetic prognostication. Many examples are given of the marked pleiotropic effect manifested by the gene or genes responsible for the syndromes. It is repeated—again and again—that the entire family or families must be studied in order to disclose all of the signs and symptoms of the specific complex. (5 figures, 24 references) Theodore M. Shapira.

Malatesta, C. **Albers-Schoenberg disease, a case report.** Boll. d'ocul. **33**:11-16, Jan., 1954.

In this 20-month-old girl, the third of three children of normal parents, the well-known skeletal and clinical changes were accompanied by mild exophthalmos and by primary atrophy of both optic nerves. Heredity was not obvious, but the first child of these parents died immediately after birth. (3 figures, 23 references) K. W. Ascher.

Meyer, E. T. **Familial ectopia lentis and its complications.** Brit. J. Ophth. **38**:163-172, March, 1954.

Familial ectopia lentis is not very rare and has been reported as part of Marfan's syndrome and as an independent anomaly. In this report studies have been carried out through four generations of a very large family in which 12 members had dislocated lenses. The question arose whether the dislocation was primary or secondary to a congenital weakness or degeneration of the zonular fibers, making dislocation a result of minor trauma. Associated with this degeneration of the fibers was liquefaction of the vitreous and herniation through the zonular defect into the anterior chamber. The subjects examined had a deep anterior chamber but nevertheless secondary glaucoma supervened in most of them and, unless both the glaucoma and the dislocated lens were surgically treated, vision was lost. Cyclodialysis was the most effective operation for the glaucoma. Whether a clear but slightly subluxated lens should be removed in order to prevent the occurrence of glaucoma later is not clear. (16 references) Morris Kaplan.

Murwitz, Paul. **Mandibulofacial dysostosis.** A.M.A. Arch. Ophth. **51**:69-72, Jan., 1954.

A case of mandibulofacial dysostosis is presented with a review of the features

of the syndrome. The oustanding features of the syndrome in this case were 1. antimongoloid palpebral fissures with colobomata of the outer portions of the lower lids and absence of the lashes in the lateral parts of the lids; 2. hypoplasia of the malar bones, rudimentary zygomatic arches, micrognathia, and receding chin; 3. malformation of the external ear; 4. microstomia, high-arched palate, speech defect, irregular disposition of the teeth, and malocclusion; 5. blind fistulae between angles of the mouth and ears; 6. atypical hair growth with tongue-like extensions; 7. frontonasal angle diminished; 8. fish-like face, and 9. mental retardation. These cases must be differentiated from craniofacial dysostosis and acrocephaly with syndactyly. Treatment consists of plastic repair of defects insofar as is possible. (3 figures, 12 references)

G. S. Tyner.

Rosehr, K. **The further course of a familial macular degeneration.** Klin. Monatsbl. f. Augenh. **124**:171-179, 1954.

Two sisters of the four siblings described in 1903 by Stargardt could be reexamined. They are now 70 and 63 years old. The disease has definitely progressed over these fifty years. Not only was the macula involved, but now also a large part of the posterior pole of the fundi. Pigment changes occurred in the choroid and a secondary optic atrophy was present. The author agrees that this disease belongs to the tapeto-retinal degenerations as a kind of inverse type of retinitis pigmentosa. (5 figures, 18 references)

Frederick C. Blodi.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Bec, Pierre. **Report of an expedition to Morocco for study.** Rev. intern. du trachome **31**:89-100, 1954.

While a resident in the ophthalmologic clinic of the University of Toulouse, the author was given the opportunity of a six weeks' study period at the Center of Ophthalmology and Experimental Trachomatology at Rabat-Salé, directed by Dr. R. Pagès, and an additional period to study trachoma in South Morocco. A description of his experiences is given, with illustrative photographs of the Center. Bec was struck by the enormity of the trachoma problem and by the severity of the accompanying bacterial infections. He reports on the progress made in therapy by the mobile field units, each unit treating up to 1,250 patients per day, and notes the effective collaboration in this treatment campaign between the Public Health Service of Morocco and the WHO and UNICEF. (4 figures)

P. Thygeson.

Habachy, Stany. **Social prophylaxis of trachoma in Egypt.** Rev. intern. du trachome **31**:235-246, 1954.

The author discusses in detail the status of trachoma in Egypt and the factors which influence its spread, particularly parasitic diseases, flies, and the acute ophthalmias. He advocates full use of social methods to combat the disease, including religion, the press, radio, and cinema. He proposes the promulgation of a law requiring notification of all cases of trachoma and the acute ophthalmias. Habachy further proposes methods to combat the plague of flies and parasitic diseases and to improve the hygiene of the peasants in their villages. (9 references)

P. Thygeson.

Hirschfelder, Max. **Ophthalmology in rural areas.** Illinois M. J. **105**:66-69, Feb., 1954.

Hirschfelder gives an interesting description of the activities of the ophthalmologist in a rural area. Standards are much higher than they were only a few decades ago. Expansion in public health measures and better training of the oph-

thalmologists have contributed to a marked reduction in the incidence of blindness. The resourceful practitioner adapts his methods of examination and treatment to the facilities which are available. Most surgical procedures can be carried out safely and successfully in small town general hospitals. Periodic refresher residencies for a few months now and again are suggested to keep the efficiency of ophthalmologists who practice away from teaching centers at peak level

F. H. Haessler.

Mackensen, G. **The poking of eyes in blind children.** Klin. Monatsbl. f. Augenh. **124**:201-202, 1954.

In a short addition to this paper (Klin. Monatsbl. f. Augenh. 122:394-402), the author reports further proof of his observation from the State School for the Blind. Frederick C. Blodi.

Pines, Noah. **Observations on the incidence of trachoma in London.** Rev. intern. du trachome **31**:153-156, 1954.

In a short note, Pines reports his observations on trachoma during 30 years of service at the Israelite Hospital in London. In his records of 20 cases he noted that the majority of patients were over 50 years of age, of Jewish origin, and born in Russia, Poland, Lithuania, and Romania where they had become infected. All were in Stages III or IV according to MacCallan's classification. No inclusions were found in any case. There was one case of unilateral trachoma which had been contracted on a visit to Austria. The author concludes that most cases of trachoma in England were the result of infection abroad but he refers to an epidemic that occurred 20 years ago in East London in the course of which he saw more than 80 cases. P. Thygeson.

Sobieska Clar, Helena. **Causes of appearance and ways of fighting scrofulous diseases of the eye in the area of Elblag, from a social and economic point of view.** Klinika Oczna **23**:273-276, 1953.

The author noticed an unusual number of patients with scrofulous diseases of the eye in the area of Elblag; the highest numbers were seen in spring and in fall. The climate and the war are the reasons for this. Elblag has very few clear sunny days during the year. During the war the area was flooded with sea water; a considerable loss of calcium from the soil resulted. Also there is too much fluorin in the drinking water. Fluorin replaces calcium in the body when present in excessive amounts. The author feels that the inadequate amount of calcium in the diet and in the organism explains the number of cases of scrofulosis and advises that appropriate measures should be taken concerning the soil, drinking water and diet. (5 references) S. Brandon.

Tacticos, Georges. **Greek legislation against trachoma and other communicable eye diseases.** Rev. intern. du trachoma **31**: 157-163, 1954.

In 1936 the first law providing for compulsory treatment of trachoma and the acute ophthalmias was promulgated in Greece. In areas particularly affected by trachoma, the law requires a prenuptial certificate stating that the parties were free from trachoma and other contagious eye diseases. In 1939 a central organization to combat trachoma was created in Athens. Later laws provided for the detection of the disease and the grouping of trachomatous children in special schools. The author states that the poor post-war economic conditions have interfered with the progress of the anti-trachoma program but that it has nevertheless already accomplished important objectives.

P. Thygeson.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at least three months before the date of occurrence.

DEATHS

Dr. Eugene Loring Bulson, Fort Wayne, Indiana, died April 13, 1954, aged 60 years.

Dr. Donald Watson Cady, Pasadena, California, died April 14, 1954, aged 60 years.

Dr. William Weir Fitzgerald, Yonkers, New York, died April 20, 1954, aged 49 years.

ANNOUNCEMENTS

NINTH PROCTOR LECTURE

Dr. Ludwig von Sallmann, director of research in ophthalmology, College of Physicians and Surgeons, Columbia University, New York, will give the ninth Francis I. Proctor Lecture on ophthalmology on Thursday evening, September 30th, at the Morrison Auditorium, Golden Gate Park, San Francisco. The subject of Dr. von Sallmann's address will be "Responses of intraocular and blood pressure to electric stimulation of the diencephalon."

PHYSIOLOGY AND PATHOLOGY COURSE

Visiting faculty for the postgraduate course in physiology and pathology of the eye sponsored by the Department of Ophthalmology, State University of Iowa College of Medicine, at Iowa City, on September 24th and 25th will be: Dr. Gunnar von Bahr, Uppsala, Sweden; Dr. Giambattista Bietti, Parma, Italy; Dr. Josef Boeck, Graz, Austria; Dr. J. H. Doggart, London; Dr. Hans Goldmann, Berne, Switzerland; Dr. Harold Henkes, Rotterdam, The Netherlands; Dr. G. B. J. Keiner, Zwolle, The Netherlands; Dr. H. K. Mueller, Bonn, Germany; Dr. Francis Heed Adler, Philadelphia; Dr. Peter C. Kronfeld, Chicago; Dr. Alfred E. Maumenee, San Francisco.

Members of the department staff are: Dr. Alson E. Braley, head; Dr. P. J. Leinfelder, Dr. Hermann M. Burian, Dr. George W. Bounds, Jr., and Dr. Frederick C. Blodi.

YALE CONFERENCES

Yale University School of Medicine in co-operation with the West Haven Veterans Administration Hospital, Hospital of St. Raphael, and Grace-New Haven Community Hospital offers postgraduate conferences in ophthalmology from January to May, 1955. Chairman of the series, as well as of the Section of Ophthalmology, is Dr. Rocko M. Fasanella. Dues of $15.00 are payable to Dr. Arthur Ebbert, Jr., assistant dean in charge of postgraduate medical education, 333 Cedar Street, New Haven, Connecticut.

The program follows:

January 7, 1955: "A procedure for cataract extraction: Routine direct zonular stripping," George Corcoran, Jr., M.D., Mercy Hospital, Springfield, Massachusetts. Followed by a panel on "Routine uncomplicated cataract extraction covering: Types of anesthesia, flap section (knife or keratome), suture (gut or silk; placement), iridectory, delivery (forceps or erisophake; tumbling or sliding)."

January 28, 1955: "Some recent advances in biomicroscopy," Milton L. Berliner, M.D., associate attending ophthalmologist and chief of clinic, New York Hospital; assistant professor of ophthalmology, Cornell; director, Department of Ophthalmology, Medical Arts Hospital and Dispensary.

February 11, 1955: "Management of reading problems in New Haven," Josephine E. Williams, M.A. reading consultant New Haven Board of Education; John Duggan, B.A. reading consultant and assistant to the dean of freshmen, Yale University, and Elva E. Knight, assistant professor of education and psychology, New Haven State Teachers College.

February 25, 1955: "An evaluation of the plastic procedures available to the ophthalmic surgeon," Arthur Gerald DeVoe, M.D., professor ophthalmology and chairman New York University-Bellevue Medical Center.

March 11, 1955: "Diagnostic features of the retinopathies," I. S. Tassman, M.D., attending surgeon, Wills Hospital; clinical professor of ophthalmology, Graduate School, Pennsylvania University; chief ophthalmology clinic, Graduate Hospital, Pennsylvania University.

March 25, 1955: (Tentative) Edwin B. Dunphy, M.D., chief, ophthalmology, Massachusetts Eye and Ear Infirmary, Massachusetts General Hospital; professor of ophthalmology, Harvard University. Subject to be announced.

April 8, 1955: Case presentations, some unusual cases of eye photography. Ernest Rosenthal, M.D., director of eye photography, Yale School of medicine.

April 29, 1955: "Comments on corneal transplantation," Ramon Castroviejo, M.D., consulting ophthalmologist, St. Clare Hospital; assistant clinical professor of ophthalmology, Columbia University College of Physicians and Surgeons; attending ophthalmologist and instructor of ophthalmology, Presbyterian Hospital, New York City.

May 13, 1955: "Common procedures in lid repair," Sidney A. Fox, M.D., assistant clinical professor of ophthalmology, New York University.

Journal club

This Journal Club is conducted by the resident staff in ophthalmology, Yale University School of Medicine, at a noon luncheon, usually on the third Wednesday of the month. The subject is usually taken from the most recent "Summary of the Year" in the *Archives of Ophthalmology*. Open to all interested.

Principles of ophthalmic surgery

One evening weekly as arranged during the calendar year, the Department of Ophthalmology, Yale University School of Medicine, offers for the resident staff in ophthalmology and a very limited enrollment of Connecticut ophthalmologists (fee, $75.00) who are expected to furnish their own instruments, a course on the principles of ophthalmic surgery. Animal eyes and a small number of cadavers will be available.

In general, supervision and subjects will include the following:

Anatomy: Cadaver available to residents. Supplemented with review in conjunction with Anatomy 202, Dr. W. U. Gardner and staff.

Surgery of lacrimal sac: J. Alexander Van Heuven, M.D., and R. M. Fasanella, M.D.

Kroenlein operation and exenteration: Andrew S. Wong, M.D.

Enucleation and allied operations: Frederick E. Mott, M.D.

Lid plastic surgery: C. C. Clarke, M.D. (utilizing cadaver).

Strabismus: C. C. Clarke, M.D. (in operating room).

Intraocular surgery (glaucoma and cataracts). Five separate sessions:

1. Sclero-iridectomy, goniotomy. Eugene M. Blake, M.D.
2. Suprachoroidal iridencleisis, Francis P. Guida, M.D.
3. Elliot trephining operation, William H. Ryder, M.D.
4. Cyclodiathermy, Frederick A. Wies, M.D.
5. Iridectomy, iridencleisis, Arthur M. Yudkin, M.D.

Intraocular foreign bodies: Arthur M. Yudkin, M.D.

Corneal transplantation: Rocko M. Fasanella, M.D.

Course on glaucoma

A course on glaucoma with particular emphasis on gonioscopy and study of the anterior angle will be given at the Brooklyn Eye and Ear Hospital on November 15, 16, and 17, 1954. Ample opportunity for practical instruction in the use of the goniolens will be given and material from the glaucoma clinic will be utilized.

The course will be given by Dr. Daniel Kravitz, assisted by Dr. Walter V. Moore, Dr. Mortimer A. Lasky, Dr. Harold F. Schilback, and Dr. Arthur Shainhouse. Registration is limited to six ophthalmologists only. Application and the fee of $40.00 may be addressed to Dr. Daniel Kravitz, Brooklyn Eye and Ear Hospital, 29 Greene Avenue, Brooklyn 38, New York.

Memphis convention

The annual eye, ear, nose, and throat convention sponsored by the Memphis Society of Ophthalmology and Otolaryngology will be held on February 5, 6, and 7, 1955, at the Peabody Hotel, Memphis. The following guest speakers will appear on the program:

Dr. George E. Shambaugh, Chicago; Dr. Paul H. Holinger, Chicago; Dr. Harold G. Scheie, Philadelphia; Dr. Frederick C. Cordes, San Francisco; Dr. F. Bruce Fralick, Ann Arbor; Dr. Dean M. Lierle, Iowa City.

Miscellaneous

Solar retinitis

The newspapers and television broadcasts gave excellent preventive advice to the observers of the eclipse seen June 30, 1954. It would be most interesting to tabulate all the cases of eclipse blindness in which medical care has been sought in order to see how well the job was done.

I would appreciate receiving short abstracts from all ophthalmologists to complete this tabulation. Credit of course will be given each contributor to this study. Isadore Givner, M.D., 108 East 66th Street, New York 21, New York.

Gonioscopic lens solutions

Sterile methylcellulose in normal saline solution, having an index of refraction which matches that of the cornea and the gonioscopic prism, is offered free of charge in 15-cc. dropper bottles to ophthalmologists and glaucoma clinics using the Allen and Goldman gonioscopic prisms routinely. The specially prepared sterile solution is for use with gonioscopic lenses and is not for therapeutic use. Requests should be addressed to Robert R. Feinstein Research Associates of 37 West 57th Street, New York, New York.

Dedication of novel

Borghild Dahl's latest novel, *Homecoming* (published by E. P. Dutton & Co., New York), which has recently been issued in talking books for the blind, has been dedicated to her two eye surgeons, Dr. William L. Benedict of Rochester, Minnesota, and Dr. Raymond L. Pfeiffer, of the Medical Center, New York City.

Societies

Pennsylvania academy

More than 350 members and guests attended the 12th annual meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology which was held recently at the Bedford Springs Hotel, Bedford, Pennsylvania.

Speakers on ophthalmology were: Dr. William B. Clark, New Orleans; Dr. George J. Dublin, Philadelphia; Capt. Frederick Harbert, Philadelphia; Dr. Charles E. Iliff, Baltimore; Col. J. H. King, Jr., Washington, D.C.; Dr. James L. McGraw, Syracuse, New York; Dr. Raymond L. Pfeiffer, New York; Dr. A. D. Ruedemann, Detroit; and Dr. William E. Krewson, III, Philadelphia.

Officers of the association are: President, Dr. James H. Delaney, Erie; president elect, Dr. William T. Hunt, Jr., Philadelphia; secretary, Dr. Daniel S. DeStio, Pittsburgh; treasurer, Dr. Bruce A. Grove, York; editor of the *Transactions*, Dr. Benjamin F. Sanders, Reading.

The 1955 meeting of the association will be held jointly with the New Jersey Ophthalmological Society and the West Virginia Eye, Ear, Nose, and Throat Society, at the Traymore Hotel, Atlantic City, New Jersey. The dates are to be announced.

Program at Oxford

On the program of the 1954 Oxford Ophthalmological Congress were:

Discussion on "The significance of macular changes," openers: Mr. Philip Jameson Evans, Mr. L. H. Savin, and Dr. Robert C. Davenport. "Fixation disparity," Mr. G. T. Willoughby Cashell; "The treatment of orbital and lid tumors," Dr. Edmund B. Spaeth, who also showed a film on "Krönlein orbitotomy for the removal of a neuroma of the optic nerve"; "The surgical treatment of ectopia lentis in the adult," Mr. O. M. Duthis; "Orbital implants," Mr. M. J. Roper Hall.

"Ocular ochronosis," Mr. S. J. H. Miller; "Assessment of visual incapacity following industrial injury," Mr. H. Campbell Orr; "A new muscle balance test," Dr. H. J. Hegner; "Facts, fallacies, and failures in cataract surgery," Sir Henry Holland; "The proprioceptive impulses from the extraocular muscles," Dr. S. Cooper, Dr. P. M. Daniel, and Prof. D. Witteridge; "Mercurialentis," Dr. J. Stewart Gourlay.

Discussion on "Postoperative ophthalmic care," openers: Mr. F. A. Williamson-Noble and Mr. J. C. Drummond Currie; "The magnetic intraocular seed implant," Dr. J. Horton Young; "Eyedrops and pyocyanea," Dr. M. Klein. Two films were shown by Mr. W. Hedley Summerskill, "Dacryocystorhinostomy by intubation" and "Scleral implantation."

Mr. Frederick Ridley, London, gave the Doyne Memorial Lecture. The subject of Mr. Ridley's address was "The contact lens in investigation and treatment."

AMA officers

The following officers were elected at the recent meeting of the Section on Ophthalmology, American Medical Association, in San Francisco: Chairman, Dr. Erling Hansen; vice chairman, Dr. Watson Gailey; secretary, Dr. Harold G. Scheie (elected at the 1953 meeting for a term of three years).

The prize in ophthalmology was presented to Dr. Otto Barkan of San Francisco for his large part in popularizing modern concepts of glaucoma; for his writings and teachings that goniotomy occupies the prominent role in the surgery of congenital glaucoma today; and for his good work in the field of ophthalmology.

Personals

Pan-American interim meeting

Dr. Murray F. McCaslin, Pittsburgh, recently back from South America, reports that:

The third interim meeting of the Pan-American Association of Ophthalmology was held in São Paulo, Brazil, June 12th to 14th. "Prevention of blindness" and "Recent advances in the therapy of eye diseases" were the themes of the program.

An ophthalmologic congress, celebrating the fourth centennial of São Paulo, was held June 9th through June 17th. In addition to the Pan-American Association of Ophthalmology, the other participating organizations were the South American Meridional Ophthalmological Society, the VIII Brazilian Congress of Ophthalmology, and the International Congress of Oto-Neuro-Ophthalmology.

Several physicians from the United States and members of the National Society for the Prevention of Blindness participated in the programs.

Instructional courses were instituted for the first time at this meeting of the Pan-American Association. The courses were well attended, and consideration is being given to their continuance at future meetings.

The ophthalmologists of São Paulo were hosts at dinner parties in their homes to groups of participants on the programs. An excursion to the port city of Santos and to an experimental coffee farm near Compinas, a native "barn dance," and the banquet at the Hotel Esplanada were the highlights of entertainment.

Elected to honorary membership

Dr. Jerome A. Hilger, Saint Paul, Minnesota, and Dr. Leonard Christensen, Portland, Oregon, have been elected to honorary membership in the Kansas City Society of Ophthalmology and Otolaryngology.

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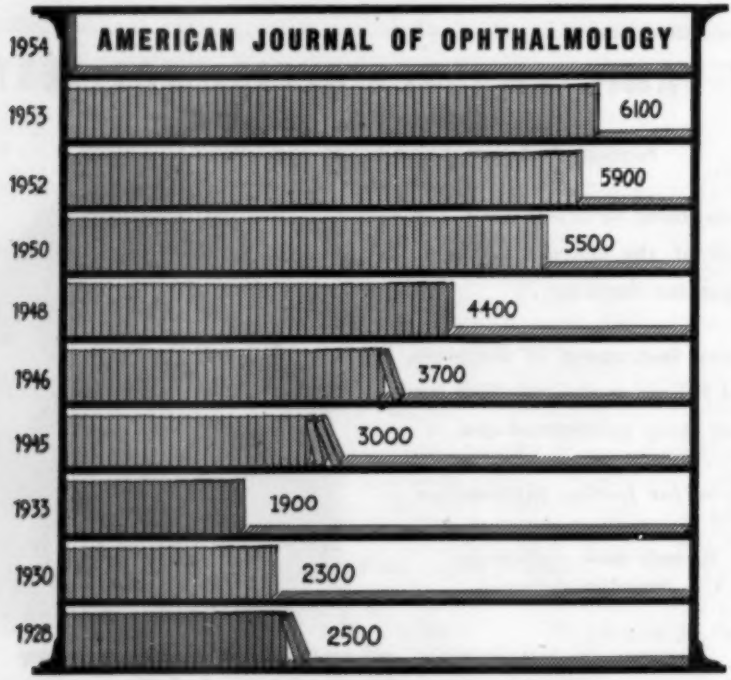

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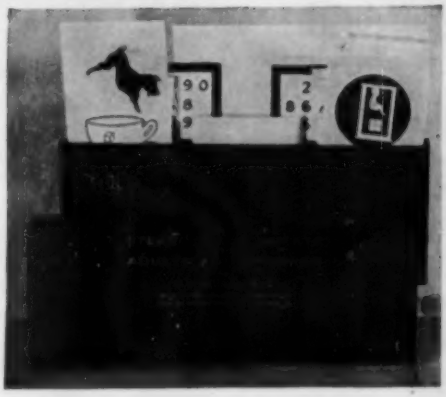

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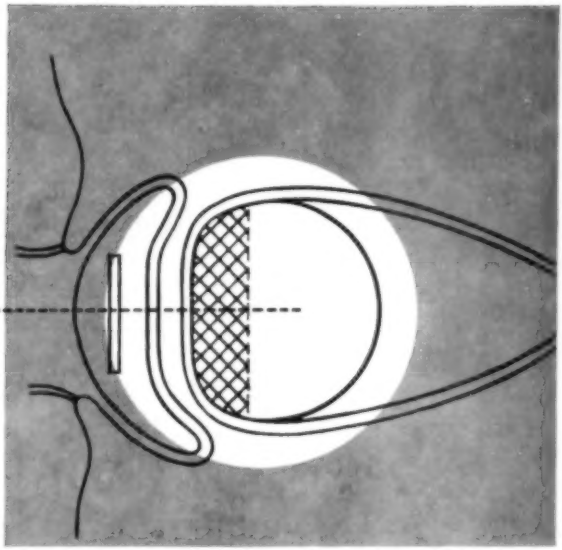

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